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THE LARYNGOSCOPE.

VOL. LX

FEBRUARY, 1950.

No. 2

ANTIBIOTICS AND SINUS INFECTIONS.*†

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A comprehensive discussion of the treatment of sinus infections would include consideration of such factors as abnormal anatomy, drainage, immunity, allergy, metabolic and endocrine deficiencies, nutrition and the use of the antibiotics. I shall limit my remarks to the latter subject.

During the past two years clinical observation has made me increasingly aware of the fact that I was not seeing as satisfactory response to the use of penicillin as I had observed two to three years ago, even though rather large doses have been used during 1949. In an effort to substantiate and explain this clinical impression, I have reviewed the charts of patients with sinus infection who have had cultures from pus in the nose made on penicillin sensitivity plates during the past five years. The information obtained was rather startling, as is shown in Table 1.

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otolological Society, Inc., New York City, N. Y., Jan. 5, 1950.

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TABLE 1.
CULTURES FROM PUS IN THE NOSE IN CASES OF ACUTE
OR CHRONIC SUPPURATIVE SINUSITIS.
BLOOD AGAR PLATES TREATED WITH PENICILLIN.

Year	Number Cultures	Gram Positive Cocci Only
1945	10	80% sensitive to 0.2 unit per cc. (inhibited by)
1946	44	82% sensitive to 0.2 unit per cc.
1947	33	60% sensitive to 0.2 unit per cc.
1948	46	26% sensitive to 0.2 unit per cc.
1949	48	12% sensitive to 0.2 unit per cc.

My interpretation of the data in Table 1 is as follows: Because of the widespread use of penicillin dust in "sniffers," penicillin nose drops, penicillin aerosol inhalations and *inadequate dosage* by mouth or by injection, the more sensitive organisms have been killed, and the more resistant strains have survived to be coughed and sneezed onto friends and relatives. Thus the bacterial flora of the upper respiratory tract has in a period of three short years become relatively resistant to the action of penicillin.

Dr. Fleming¹ said in 1946, "It is to be hoped that penicillin will not be abused as were the sulfonamides. . . . It is the administration of too small doses which leads to the production of resistant strains of bacteria, so the rule in penicillin treatment should be to give enough."

Pus for these cultures was obtained from the interior of the nose by means of the suction collector shown in Fig. 1. The organisms demonstrated were various types of staphylococci, streptococci and pneumococci, grown aerobically on blood agar plates treated with various concentrations of penicillin, with one untreated control plate. I am not stating that these cultures represent the infecting organism, the pathogen solely responsible for the disease, but they do represent the

bacterial flora found in pus in the noses of patients having obvious suppurative sinus infection, and show the decreasing sensitivity of these organisms to the action of penicillin.

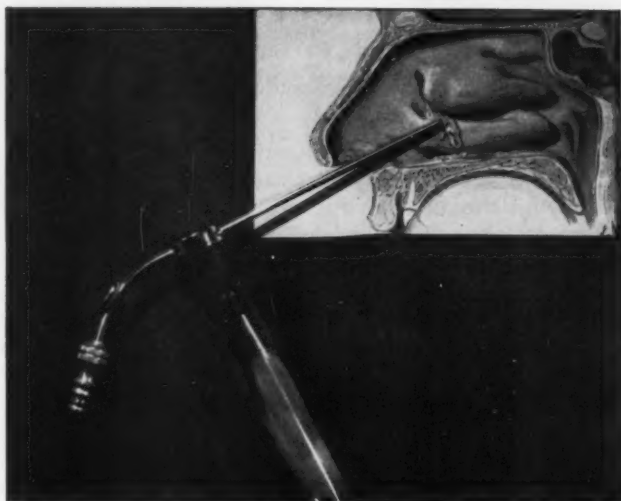


Fig. 1. A suction-collector obtaining an uncontaminated specimen of pus from the nose for smears and cultures.

This study has at least one serious flaw: no anaerobic cultures were made. The preparation of this paper made me find some articles that I had previously overlooked. Those written by Galloway,² Williams and Heilman,³ Pastore and Williams⁴ impressed upon me the importance of the micro-aerophilic streptococci as causal agents of sinus infections and their complications.

My belief that the gram positive cocci demonstrated by culture are at least some of the pathogens is strengthened by the smears which show many cocci mixed with the neutrophils. Quite often two organisms, usually a staphylococcus and a streptococcus, were found in a given sample of pus, and their behavior with respect to penicillin sensitivity plates varied considerably, as is shown by Table 2.

TABLE 2.
CULTURE ON PENICILLIN TREATED BLOOD AGAR PLATES.

	0.2	0.5	1.0 Units Per Cc.
Staph. aureus	4+	4+	4+
Strep. viridans.....	1+	0	0

+ equals growth.

0 equals no growth.

This method of testing bacterial sensitivity has also been used by Nichols and Needham.⁵

In 1947, I wrote a paper,⁶ based on cases treated during 1946, indicating that nearly all patients with acute, and some patients with chronic suppurative sinusitis could be cured by intramuscular injection of penicillin, using 50,000 units every three hours, day and night, for a period of seven days, *providing the infection was due to organisms sensitive to penicillin in concentration of 0.2 unit per cc.* This is perhaps still true, but only 12 per cent of the organisms cultured from pus in the nose are at present writing sensitive to this concentration (at least in Central Pennsylvania).

The frequency of penicillin-resistant infections is often not appreciated, and I have seen the diagnosis of "Virus X" infection used more than once to explain the cause of a penicillin-resistant infection. Barber⁷ and her co-worker in England reported an incidence of 14.1 per cent resistant strains of staph. pyogenes in 1946 with strains resistant to 10 units, increasing to 38 per cent in 1947, and to 59 per cent in 1948. Nichols and Needham,⁵ in 1949, reported that 68 per cent of 50 strains of staph. aureus were resistant to 1.6 units of penicillin.

TABLE 3.
1949 PENCILLIN SENSITIVITY CULTURES FROM PUS
IN THE NOSE.

Number Cultures	Concentration of Penicillin in Units Per Cc.	Per Cent of Organisms Inhibited
48	0.2	12%
65	0.5	43%
65	1.0	55%
21	3.0	81%

The data in Table 3 suggest that blood levels in 1949 have to be 15 times as high as in 1946 in order to inhibit the growth of 81 per cent of the gram positive cocci found in pus in the nose.

These data cast doubt on the current idea that an intramuscular injection of three or four hundred thousand units every 24 hours will be effective as treatment for most respiratory infections, and make ludicrous the reference to 0.03 unit as the "minimal effective therapeutic level."

Even the pneumococcus has become more resistant, so that in 1949 it frequently grows on plates containing penicillin 0.5 unit per cc., as shown in Table 4.

TABLE 4.
SENSITIVITY CULTURES FROM PUS IN THE NOSE.

	Penicillin			Dihydro-Streptomycin			Aureomycin		
	0.2	0.5	1.0	5	10	25	1	2	5
Staph. aureus	2+	2+	2+	1+	1+	1+	1+	0	0
Dip. pneumoniae	3+	1+	0	0	0	0	0	0	0

+ equals growth.

0 equals no growth.

The following quotation from Sir Alexander Fleming's book^s entitled, "Penicillin, Its Practical Application," should serve to keep our thinking straight: "There are a few simple rules for the use of penicillin in treatment of bacterial infections. These may seem too simple — even childish — but experience in watching penicillin being used has made it clear that these rules are often broken with resulting disappointment.

"1. It should be used only when there is an infection by a penicillin-sensitive microbe.

"2. Penicillin must be administered in such a way that it comes in contact with the infecting microbe.

"3. The dose should be such that in the infected area the concentration of penicillin is sufficient to destroy the bacteria.

"4. The treatment should be persisted in until the infection is defeated."

Regardless of the numerous articles advocating the topical use of penicillin as nose drops, aerosol inhalations, by injection into antra and by displacement, the words of Dr. Fleming have led me to ignore these methods because they do not live up to his four cardinal principles. Since penicillin must be administered in such a way that it comes in contact with the infecting microbe, and because the germs are located at various depths *within* the tissues where they can be reached only by blood-borne penicillin, I use *only intramuscular injections* in the treatment of sinus infections.

It is not enough to say "penicillin-sensitive." For intelligent treatment we must know *how* sensitive the organism is if we are going to be able to determine when to use penicillin and *how much to use*. The following quotation from Marvin⁹ indicates how misleading hasty conclusions may be: "Loewe deserves great credit for having demonstrated the value of

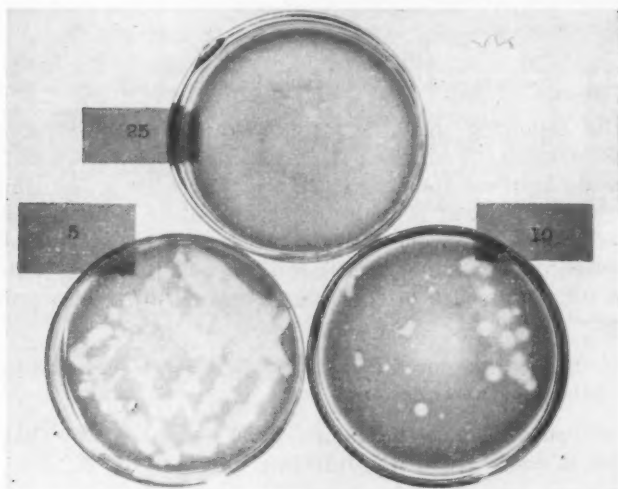


Fig. 2. The growth of a hemolytic streptococcus on blood agar plates containing 5, 10 and 25 micrograms of dihydro-streptomycin per cc. Note complete inhibition of growth on the 25 microgram plate.

large doses of penicillin for subacute bacterial endocarditis after the Penicillin Committee had officially declared it to be of no value." In some cases of sinus infection I use 300,000 units of procaine penicillin with 100,000 units of soluble penicillin every three hours, when smaller doses fail to clear up the infection, and I prefer the aqueous suspension. I think this will maintain a serum concentration of 3.0 units or more, but I have no exact data.

In choosing a chemotherapeutic agent, *the size of the dose* and the *duration of treatment* are both of importance. The site of the infection as well as the degree of penicillin-sensitivity of the organism will partially determine the duration of treatment as is indicated by Table 5 taken from Dr. Fleming's book,⁸ published in 1946.

TABLE 5.

AVERAGE MINIMUM DURATION OF DAYS OF TREATMENT FOR PATIENTS RECEIVING 60,000 UNITS EVERY THREE HOURS.

Uncomplicated gonorrhea	1 day
Vincent's angina	2 days
Erysipelas, impetigo	3 days
Carbuncles, cellulitis.....	5 days
Breast abscesses.....	7 days
Otitis media and mastoiditis.....	8 to 10 days
<i>Sinusitis</i>	10 to 12 days
Complicated septicemias	* 12 to 21 days
Endocarditis	21 to 28 days

Our therapy should not be dictated by the size of the dose marketed in 1 cc. ampules and alleged to be adequate daily dose for most infections. The 96-hour advertising has also resulted in much undertreatment.

When "Penicillin-resistant" infections are encountered, one naturally thinks of other antibiotics. We started making cultures on streptomycin-treated plates in October, 1947, using 5, 10 and 25 micrograms per cc. Fig. 2 shows the appearance of these plates. As soon as dihydro-streptomycin became

available, sensitivity cultures were made with this antibiotic and we found that its antibacterial effect with reference to the cocci was the same as that of streptomycin in the same concentration.

TABLE 6.
1949 DIHYDRO-STREPTOMYCIN SENSITIVITY CULTURES
FROM PUS IN THE NOSE.

Number Cultures	Concentration of Dihydro-Streptomycin in Micrograms Per Cc.	Per Cent Inhibited
65	5	66%
65	10	86%
65	25	93%

Table 6 shows that 86 per cent of the cocci are inhibited by 10 micrograms of dihydro-streptomycin per cc. blood agar. Thompsett¹⁰ has shown that serum concentrations of this amount of streptomycin can be produced by giving 10 milligrams per kilogram body weight every 12 hours. This means that an effective dose for a 150-pound person is 0.7 gm. every 12 hours, and according to his data this dose should seldom, if ever, produce toxic reactions; however, Thompsett was reporting only the toxic effects of streptomycin. Recent information obtained from Dr. Glorig¹¹ indicates that dihydro-streptomycin can have a *delayed* toxic effect on the cochlear division of the VIIIth nerve. Thirteen per cent of his patients given 2 gm. of dihydro-streptomycin daily for at least 40 days had severe hearing loss. I have seen one patient who received only 22.6 gm. of dihydro-streptomycin who developed hearing loss and severe persistent tinnitus two months later; hence, we must be cautious in administration of dihydro-streptomycin and be certain that renal function is not impaired.

This is unfortunate because our cultures and clinical observations indicate that at present dihydro-streptomycin is much more effective than penicillin in usual dose.

The results of our aureomycin sensitivity cultures are shown in Table 7.

TABLE 7.
1949 AUREOMYCIN SENSITIVITY CULTURES MADE
FROM PUS IN THE NOSE.

Number Cultures	Concentration of Aureomycin in Micrograms Per Cc.	Per Cent Inhibited
57	1	86%
57	2	95%
57	5	98%

Herrell and Heilman¹² state that a serum concentration between 2 and 4 micrograms can be maintained by giving 750 mg. of aureomycin by mouth every six hours; therefore, I have used this dose and interval when aureomycin appeared to be the drug of choice. It is expensive and may produce nausea, vomiting and diarrhea, so there are some relative contraindications to its use.

The majority of our cultures indicate that chloromycetin is *not* so effective as aureomycin against the gram positive cocci. Table 8 is an example.

TABLE 8.
SENSITIVITY CULTURES FROM PUS IN THE NOSE.

	Penicillin			Dihydro-Streptomycin		
	0.2	0.5	1.0	5	10	25
Staph. aureus	4+	4+	4+	0	0	0
Strep. viridans	4+	4+	1+	1+	0	0
	Aureomycin			Chloromycetin		
	1	2	5	1	2	5
Staph. aureus	1+	0	0	4+	3+	3+
Strep. viridans	4+	4+	1+	4+	4+	0

Table 8 also indicates how sensitivity cultures can help one decide which antibiotic to use. Because of its cost and poor antibacterial effect with reference to the gram positive cocci, I have not used chloromycetin.

I no longer use sulfadiazine except for meningitis or H. influenzae infections. The superiority of penicillin over sulfa-

diazine was shown by Wilcox¹³ and in 42 per cent of his cases the use of sulfadiazine was discontinued because of toxic symptoms or failure to cure the infection.

DISCUSSION.

I have no statistical data to present showing results of treatment with penicillin alone, because most patients received dihydro-streptomycin as well as large doses of penicillin. The doses I now use for a 150-pound person are procaine penicillin, G 300,000 U combined with 100,000 U crystalline penicillin every three or every six hours, day and night. I subscribe to Dr. Fleming's statement that maximal antibiotic activity is dependent upon high sustained serum concentrations. In addition, many patients were given dihydro-streptomycin 0.7 gm. every 12 hours and treatment was continued for seven to 10 days. This treatment program has cleared up some long standing chronic infections without operative treatment other than puncture and lavage of pus filled antra. Other patients with more extensive disease required judicious but minimal surgery.

CONCLUSIONS.

Most of the gram positive cocci found in the nose are at present (1949) relatively insensitive to the action of penicillin. Penicillin is still effective in treatment of sinus infections if doses as large as 1.6 to 3.2 million units are given daily for a period of seven to 10 days. Sensitivity cultures on blood agar plates are a useful method of determining which antibiotic to use and the size of the dose to be used.

BIBLIOGRAPHY.

1. FLEMING, A.: *Chemotherapy, Yesterday, Today and Tomorrow*. Cambridge University Press, 1946.
2. GALLOWAY, T. C.: Infections with Anaerobic Streptococci with Special Reference to Cranial Osteomyelitis. *Tran. Amer. Laryngol. Assn.*, 64:35, 1942.
3. WILLIAMS, H. L., and HEILMAN, F. R.: Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus. *Arch. Otol.*, 25:196, 1937.

4. PASTORE, P. N., and WILLIAMS, H. L.: Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus. *Proc. Staff Meet. Mayo Clin.*, 13:7, Jan. 5, 1938.
5. NICHOLS, D. R., and NEEDHAM, G. M. Aureomycin in the Treatment of Penicillin-Resistant Staphylococci Bacteremia. *Proc. Staff Meet. Mayo Clin.*, 24:157, Mar. 30, 1949.
6. DAVISON, F. W.: Penicillin Treatment of Sinus Infection. *Pa. Med. Jour.*, 51:272, Dec., 1947.
7. BARBER, M., and ROZWADOWSKA-DOWZENKO, M.: Infection by Penicillin-Resistant Staphylococci. *Lancet*, 2:641, Oct. 23, 1948.
8. FLEMING, A.: Penicillin, Its Practical Application. Philadelphia: The Blakiston Co., 1946.
9. MARVIN, H. M.: Recent Advances in the Field of Cardiovascular Disease. *Bull. N. Y. Acad. Med.*, Nov., 1948.
10. THOMPSETT, R.: Relation of Dosage to Streptomycin Toxicity. *Tran. Amer. Laryngol., Rhinol. and Otol. Soc.*, p. 54, 1948.
11. GLORIG, A.: A personal communication.
12. HERBELL, W. E., and HEILMAN, F. R.: Aureomycin Studies on Absorption, Diffusion and Excretion. *Proc. Staff Meet. Mayo Clin.*, 24:157, Mar. 30, 1949.
13. WILCOX, J. G.: Penicillin Treatment of Acute Middle Ear and Mastoid Infections. *Pa. Med. Jour.*, 50:574, Mar., 1947.

THE SURGICAL TREATMENT OF BILATERAL POSTICUS PARALYSIS OF THE LARYNX.*

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Prior to King's report on the treatment of bilateral paralysis of the larynx by transposition of the omohyoid muscle to the arytenoid cartilage, the surgical results were generally unsatisfactory. While the immediate effects often were favorable, after fibrosis occurred one was confronted with cicatricial as well as paralytic stenosis.

My first attempt at transposition of the omohyoid to the arytenoid cartilage was made in 1940, employing the technique described by King.¹ It proved to be only partially successful, and I attributed this to failure to shorten the omohyoid muscle sufficiently. Later the procedure was repeated on the opposite side and a satisfactory airway was secured.

In one of the early cases I could not identify the omohyoid muscle which had been incised when the thyroidectomy was done. After mobilizing the arytenoid cartilage it was secured with chromic catgut sutures to the posterior border of the wing of the thyroid cartilage. The result in this case was the best I ever had secured, and following this experience I no longer transposed the omohyoid muscle but secured the mobilized arytenoid cartilage with a silk suture passed through a drilled hole made near the posterior border of the corresponding wing of the thyroid cartilage. Motion pictures of the larynx made before and after operation in a number of these cases revealed conclusively that the arytenoid cartilage and

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vocal cord on the operated side remained immobile and the only movement noted during phonation occurred on the unoperated side. This observation also had been made by others and it now is generally believed that the success of this plan of surgical treatment is dependent upon free mobilization of the arytenoid and its lateral fixation, and that the rôle of the omohyoid muscle is of little or no importance.

In 1941, Kelly² reported his results by arytenoidectomy through a resected window in the ala of the thyroid cartilage and fixation of the cord. McCall and Gardner³ recommended transillumination of the larynx to aid in locating the position of the arytenoid and, also, the employment of a suture to bring the arytenoid through the resected window. Orton⁴ advised removal of the posterior portion of the wing of the thyroid cartilage to facilitate the approach to the arytenoid cartilage. Woodman⁵ recommended partial arytenoidectomy with retention of the vocal process and fixation of the vocal cord to the inferior cornua of the thyroid cartilage. Thornell⁶ reported an intralaryngeal approach for arytenoidectomy in bilateral abductor paralysis and reported his results in three cases. The most recent report on this subject was presented by Georges Perello,⁷ who described a new technique which consists of making a window in the thyroid cartilage and suturing the superior end of the sternothyroid muscle to the internal fasciculus of the thyroarytenoideus muscle or to the true vocal cord.

Since I have had no personal experience with the latter procedures, no further comment shall be made other than to state that the advocates of these have reported satisfactory results. I have employed the King technique with certain modifications in all my cases. In a report⁸ in 1947, based on 32 operated cases, four were unsuccessful after the first operation, but in three of these a satisfactory result was secured following operation on the opposite side. The remaining case, a teacher, refused a second operation, as she preferred to use a valved tracheal cannula and retain a good speaking voice. In these four, the original King technique in which the omohyoid muscle was transposed was used. I am in accord with Dr. King that this method is not satisfactory.⁹

Technique: In patients wearing a tracheal cannula one may insert an intratracheal tube preoperatively through the tracheotomy fistula, thus keeping the anesthetist out of the field of operation and diminishing the dangers of wound contamination. If no tracheotomy has been performed, one either can insert an intratracheal airway through the larynx preoperatively and perform tracheotomy at the termination of the operation or the tracheotomy may be performed preoperatively and an airway inserted through the tracheal stoma. While placement of an airway in the larynx often facilitates localization of the arytenoid, I believe that it increases the likelihood of injury to the mucosa when separating the capsule of the joint on its mesial aspect and also interferes with direct laryngoscopy as practiced by some surgeons to determine the width of the glottic chink at the time of operation.

While the choice of preoperative medication and anesthesia must be left to the individual operator, it is, nevertheless, imperative that no preliminary sedation or general anesthesia is employed in a dyspneic patient who has not been tracheotomized. Tracheotomy should always be done under local anesthesia without sedation. Local anesthesia has been employed in several cases for mobilization but has not been satisfactory, and either inhalation anesthesia or sodium pentothal are preferable.

A horizontal incision two inches long conforming to the skin creases of the neck is made over the wing of the thyroid so that it crosses the posterior border of the cartilage slightly below its middle. The omohyoid may be retracted or incised. The posterior border of the thyroid cartilage is identified and the inferior constrictor is incised along its attachment to the cartilage. The palpating finger should now identify the signet of the cricoid along its upper margin. As a rule, the arytenoid cannot be palpated due to its forward displacement.

The pharyngeal mucosa is separated for a short distance from the signet of the cricoid cartilage towards the midline and the posterior cricoarytenoid muscle is identified and incised along its attachment to the muscular process. As a rule there is little difficulty in identifying and finding the

muscular process of the arytenoid. I have found a small hook valuable to secure the arytenoid during these manipulations. In order to secure satisfactory mobilization it is well to separate all of the posterior cricoarytenoid muscle from the muscular process, to incise the fibres of the arytenoideus muscle as much as one can along the body of the arytenoid and then to pull the arytenoid backward to separate some fibres of the lateral cricoarytenoid muscle. The cricoarytenoid joint is opened and its attachments are incised. Care should be exercised to avoid injury to the laryngeal mucosa, particularly when separating the mesial attachment of the capsule. A blunt elevator has been found satisfactory for this procedure.

Before proceeding with the placement of the suture it is necessary to assure oneself that the arytenoid is freely mobilized. It should be possible with the aid of the hook to draw the arytenoid laterally and posteriorly so that it can be brought in contact with the posterior border of the thyroid cartilage.

Lateral fixation is accomplished by securing the arytenoid to the posterior border of the thyroid cartilage. For this purpose a full curved needle with a braided No. 0 silk suture is passed around the cartilage and secured through a hole made with a dental burr near the posterior border of the thyroid cartilage. This gives better fixation than if fascia or muscle is used. The suture should be placed around the arytenoid near its base, beneath the mucosa, passing the needle from within outward. Occasionally it is difficult to place the suture in proper position about the arytenoid since this cartilage is pyramidal in shape and the mucosa along its inner surface is in close relation to the perichondrium. In difficult cases the mucosa overlying the posterior and particularly over a part of the inner aspect of the arytenoid should be separated. Perforation of the overlying mucosa may lead to the formation of a granuloma or to infection of the wound. While it is desirable to pass the needle around the cartilage, I am sure that in many instances it is passed through a part of the cartilage along its inner aspect.

When drilling the hole for the suture one should try to maintain the arytenoid on its normal horizontal plane. If the opening is made higher so that the arytenoid is elevated when secured with the suture the airway will be correspondingly increased, but this also will produce increased impairment of the voice. The inferior constrictor muscle is then repaired and the wound closed, preferably inserting a rubber tissue drain which should be left *in situ* 48 hours.

Variations in Technique: It is difficult to secure an adequate exposure of the arytenoid cartilage without resection of the posterior part of the thyroid cartilage. Retraction of the wing of the thyroid may be facilitated by separation of the cricothyroid articulation or by cutting the inferior cornua of the thyroid cartilage with shears. This has been done on several occasions with no apparent untoward effects. Counter-pressure on the posterior border of the opposite wing of the thyroid cartilage often gives a better exposure.

In the average case, if the arytenoid is brought in contact with the inner aspect of the thyroid cartilage along its posterior border the glottis will be adequate. In two instances, in cases of paralysis occurring in men, it seemed desirable to provide a wider glottis than one secures by this method and a small notch was made in the posterior border of the thyroid cartilage so that the arytenoid could be hinged around it. In an occasional case a second suture has been placed about the arytenoid to aid in its lateral displacement.

In earlier cases I routinely performed direct laryngoscopy before securing the arytenoid to determine if an adequate glottis was secured. Too often, however, it was difficult to evaluate the findings as the patient either was lightly anesthetized or edematous changes had occurred submucosally about the field of operation and one could not be certain regarding the width of the glottic chink. I now secure the arytenoid as previously noted without reference to the size of the glottis and, with two exceptions, the airway has been adequate. In one case the separation was too great and the patient has only whisper voice. In the other, now two

months after operation, the airway does not appear wide enough and decannulation has not been carried out. A glottic chink should be approximately one-half cm. in width at its posterior end to insure an adequate airway.

End-Results: It is impossible to restore normal laryngeal function in a case of bilateral abductor paralysis. In order to improve the airway one must widen the glottis and this obviously will detract from the patient's ability to phonate. One, therefore, can state positively that the wider the glottis is, the poorer the voice will be. It has been observed that the operated side becomes fixed, and any movement that is observed occurs on the unoperated side; therefore, while one may operate on both arytenoids a unilateral operation is preferable from the standpoint of phonation. Better phonatory results also will be obtained if the arytenoid is retracted laterally without rotating it so that a tense straight-edged vocal cord will be secured.

I had previously believed that such adduction as occurred on the unoperated side could be explained by the action of the cricothyroid muscle which primarily is a tensor of the cord but also exerts some adductor action. When the cricoid cartilage is drawn upward towards the thyroid cartilage by the cricothyroid muscle, there apparently is some tilting inward of the corresponding arytenoid, which probably is a passive action. The thyroarytenoideus muscle by reason of its attachment to the vocal process also may act in a passive manner. There is considerable question whether the action of the cricothyroid muscle is of great significance in posticus paralysis. The arytenoid which is tilted forward lacks normal fixation and this interferes with the tensor action of the cricothyroid muscle. It seems reasonable that the unopposed lateral cricoarytenoid muscle may be responsible for slight adductor action on the nonoperated side. I believe that many of these patients use the extrinsic muscles which aid in narrowing the airway during phonation. In addition one must take into account that there is a change in the upper orifice of the larynx, for the epiglottis usually is tilted backward and on phonation this is increased contrary to findings in the normal larynx.

Voice Changes: In none of my operated cases has there been any material improvement in the voice. In practically all of the cases of paralysis following thyroidectomy the voice was quite clear before operation although there was breathlessness. In a majority the voice has been reasonably good following mobilization; there has been but one exception and in this the voice was a loud whisper. Patients who have worn a tracheal cannula for relief of dyspnea or have lived a precarious existence because of shortness of breath invariably are extremely grateful when the airway is increased. I have never observed a single instance in which the patient regretted having had the operation performed in spite of the voice impairment.

Statistical Data: To date I have operated upon 66 patients. In the first three and in Case 18 a second operation was necessary because the airway was not adequate. In the three the omohyoid was transposed and in the latter case there was edema of the arytenoid on the operated side which persisted. While there was no wound infection, there undoubtedly was some chondritis of the arytenoid cartilage and a second operation was performed with very satisfactory respiratory results and fair phonatory results considering that both arytenoids are immobile.

Sex: There were 56 women, nine men, and one boy aged 14 years.

Etiology: In 60 the paralysis followed one or more thyroidectomies. In three the cause was not known, but apparently was toxic or viral in origin. In one of these, a boy aged 14 years, bilateral paralysis followed measles seven years previously. In two there was trauma due to gunshot and shrapnel wounds, and in one there was bilateral paralysis following pulmonary tuberculosis.

Duration of Paralysis: In 34 cases the paralysis was of less than five years' duration. In 11 it was from five to 10 years; in 13, from 10 to 20 years, and in six it was over 20 years, the longest being $24\frac{1}{2}$ years.

Age: The patients' ages varied from 14 years, the case of the boy with measles, to 76 years. Over one-half of the patients were more than 35 years of age. Twenty-eight at some time had worn a cannula; one wore a cannula for 16 and another for 17 years.

Decannulation Following Mobilization: In the earlier cases decannulation was delayed a number of weeks. Now, however, it is carried out more promptly as there has been opportunity to observe the postoperative reactions. This also may be due to the employment of antibiotics or to more careful handling of tissues. In 20 of the last 21 cases decannulation was completed within two weeks, the earliest being five days postoperatively. In one case the tube was not removed until after three months. This patient developed a small granuloma in the posterior commissure on the operated side that suggested a contact ulcer although it probably was the result of trauma to the mucosa. Removal of the granuloma was followed by complete recovery and decannulation with a reasonably good voice.

In two cases following thyroidectomy and in one post-traumatic case the cannula has not been removed. In one of these, Case 2, there was no lateral fixation of the arytenoid as the omohyoid was employed. The patient did not wish a second operation. In the second case, now two months post-operative, the glottis appears too narrow, but there was considerable submucosal hemorrhage at the time of operation. In the post-traumatic case decannulation was carried out, but because of extensive cicatricial changes subglottically in the posterior commissure there was recurrence of the stenosis, and tracheotomy again became necessary. This case was not suitable for this type of operative procedure.

Wound Infection: Infection of the wound due to injuries to the laryngeal mucosa occurred in five of the earlier cases. In only one was this protracted, but an ultimate satisfactory result was obtained in all.

While a majority of cases of posticus paralysis result from thyroidectomy, occasionally trauma to the neck may result in

paralytic and cicatricial stenosis. It is not always easy to distinguish between these, nor can one determine preoperatively whether a satisfactory result may be secured.

Post-Traumatic Cases: In two cases, both veterans of World War II, there was a history of gunshot and shrapnel wounds of the neck and larynx with bilateral fixation of the arytenoids in the midline. The appearances were those one observes in post-thyroidectomy paralysis. In both, mobilization and lateral fixation of an arytenoid was carried out. The findings were quite unlike those observed following thyroidectomy in that there was fibrous ankylosis of the cricoarytenoid joint in one and bone ankylosis in another. The latter case, which was done more than 18 months ago, has retained an excellent airway and has been decannulated, but the patient with fibrous ankylosis had a recurrence of dyspnea about six months following decannulation and it was necessary to do a tracheotomy. On further inspection it was found that this patient had extensive cicatricial changes in the interarytenoid area, extending subglottically, which were not observed preoperatively and his case was not a suitable one for this type of operative procedure.

In conclusion, I believe that mobilization and lateral fixation of an arytenoid gives excellent results in bilateral abductor paralysis of the larynx. Other techniques have given similar results in the hands of various operators. The fundamental principles require separation of the cricoarytenoid attachments so that the vocal cord can be abducted and fixed in that position.

BIBLIOGRAPHY.

1. KING, B. T.: A New and Function Restoring Operation for Bilateral Abductor Cord Paralysis. *Jour. A. M. A.*, 112:814, 1939.
2. KELLY, J. D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles. *Arch. Otolaryngol.*, 33:293, 1941.
3. MCCALL, J. W., and GARDNER, F. S.: A Simplified Operation for Bilateral Abductor Paralysis. *THE LARYNGOSCOPE*, 53:307, 1943.
4. ORTON, H. B.: Surgical Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Larynx. *THE LARYNGOSCOPE*, 53:709, 1943.

5. WOODMAN, D.: A Modification of the Extralaryngeal Approach to Arytenoidectomy for Bilateral Abductor Paralysis. *Arch. Otolaryngol.*, 43:63, 1946.

6. THORNELL, W. C.: A New Intralaryngeal Approach in Arytenoidectomy in Bilateral Abductor Paralysis of the Vocal Cords; Report of Three Cases. *Arch. Otolaryngol.*, 50:634, Nov., 1949.

7. PERELLO, G.: New Technique for the Treatment of Paralysis of the Posterior Cricoid. *Rev. de Laryngol., Otol. and Rhinol.*, 2:80, Jan.-Feb., 1949.

8. CLERF, L. H.: The Surgical Treatment of Paralytic Stenosis of the Larynx. *Plas. and Recon. Surg.*, 2:326, July, 1947.

9. KING, B. T.: Progress in Dealing with Vocal Cord Paralysis. *Jour. Int. Coll. Surg.*, 12:288, May-June, 1949.

DEAFNESS DUE TO ALLERGY.*

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The purpose of this paper is to discuss allergy as an etiologic factor in deafness. No controversial comments are intended concerning the scientific accuracy or the physiology of allergy. The term is herein used in its broadest application.

One of the common middle ear conditions causing mild deafness is known by two popular terms: chronic secretory otitis media and chronic otitis media with effusion. Allergy was found to be the etiology in 85 per cent of 164 consecutive cases with this diagnosis. Although scientific proof that allergy affects the mucosa of the middle ear and Eustachian tube is lacking, clinical evidence indicates this lining membrane reacts similarly to that of the nose and nasopharynx. It is not conceivable that negative pressure caused by Eustachian tubal block can entirely account for the large amount of serous fluid frequently recovered from the ear and mastoid cavity following paracentesis and inflation. Direct smears of the fluid have shown scattered eosinophiles and leucocytes, but not in sufficient numbers to prove a diagnosis of allergy by present cytologic standards. Ashley¹ reports an increase in eosinophiles in material taken 1 cm. distal to the tube orifice. Dean,² Jones,³ Koch⁴ and others agree that allergy does occur in the middle ear. If this phenomenon exists, certain changes and complications occurring in the middle ear can be explained on this basis.

One year following the relief of chronic secretory otitis media, three patients had labyrinthine fenestrations for otosclerosis. Prior to surgery retraction of Shrapnell's mem-

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brane was noted in these cases. During surgery delicate elastic vascular adhesions were found, extending from that portion of tympanic membrane traversed by the chorda tympani to the medial wall of the middle ear cavity. The medial attachment was along the Fallopian canal and the canal of the tensor tympani muscle forming a membranous partition which separated the attic and middle ear cavities. As contended by Day,² this interruption of the normal aeration of the attic and mastoid cells results in the creation of a negative pressure and consequent retraction of Shrapnell's membrane. This observation, illustrated in Fig. 1, is the first step in the formation of attic cholesteatoma.

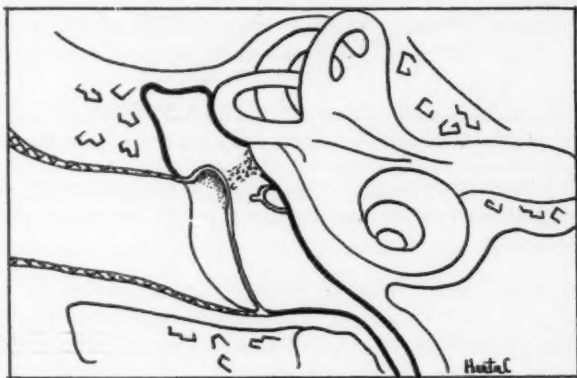


Fig. 1. Illustrates the first step in the formation of attic cholesteatoma.

The second step is the rupture of this epitympanic invagination. Clinical observations indicate that this rupture occurs as the result of two opposing forces. The exertion of an inward force on Shrapnell's membrane as the result of a negative pressure in the attic and the increased tension on the membrane by an outward displacement of the short process and head of the malleus. When the drum membrane is retracted as the result of Eustachian tubal block, the long process of the malleus is drawn inward. This pushes the short process and head of the malleus outward, increasing the tension on Shrapnell's membrane.

The walls of the ruptured pouch become adherent to the attic walls, replacing mucous membrane with skin. Stage 3 is illustrated in Fig. 2.

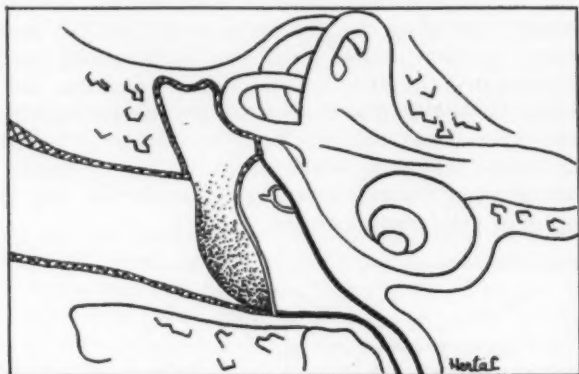


Fig. 2. Illustrates the third step in the formation of attic cholesteatoma.

The fourth stage occurs when there is an inadequate outlet for desquamated epithelium resulting in the formation of attic cholesteatoma.

The progression from a simple chronic secretory otitis media to a perforation of Shrapnell's membrane has been observed in three cases. One of these cases, illustrated below, developed an attic cholesteatoma.

A seven-year-old girl was first seen in our office in 1942 with a history of mild deafness beginning at three years of age. Upon examination a bilateral secretory otitis media with fixed depressions of Shrapnell's membrane was found. Treatment was instituted with a more favorable response occurring in the left ear. Several recurrences responded to therapy until 1946, when the patient discontinued treatment. One year later the patient returned following an acute nasopharyngitis. A soft polypoid mass was found protruding through a perforation of Shrapnell's membrane in the right ear. Following removal of the polyp and local treatment, the right ear became dry. Four months later, after an acute sore throat, attic perforations with discharge were found in both ears. Under treatment the attic perforation in the left ear healed and has not reopened in the past two years. The right ear progressed to an active attic cholesteatoma requiring a radical mastoidectomy in 1949. The total time for this transition was 11 years. As a point of interest, the number of cases of chronic attic suppurations with cholesteatoma does not seem to be affected by the current use of antibiotics.

During the first six weeks after operation, six fenestration cases developed an exacerbation of an existing allergy. Simultaneously a slit-like opening appeared along the most anterior edge of the flap through which a mucoserous fluid discharged into the operative cavity in direct proportion to the allergic state. Since the middle ear cavity remained free of fluid, it was supposed this slit-like opening was a sealed channel to the Eustachian tube. All cavities epithelized, and all cases reached a practical hearing level. Allergic management was instituted in all cases. Three became dry after an average of 15 months. The remaining cases continued to have intermittent wet cavities, one case over a period of six years. This condition did not occur in 24 fenestration cases in which the presence of allergy was recognized prior to surgery and some allergic management instituted.

A similar reaction was observed in radical mastoid cavities. A review of 89 consecutive radical mastoidectomies over a period of four years revealed 17 cases still had intermittent or continuous wet cavities. Ten of these 17 cases were found to have nasal allergy. Of these cases, all but three have dry cavities following allergic management. The source of the moisture was again tubal and entered the mastoid cavity either through an opening adjacent to the anterior wall of the epitympanum or some portion of the remaining drum.

The relationship between allergy and the amount and character of discharge in patients with chronic central perforation of the drum has been noted. Findings in this problem concur with Dohlman's⁴ report "that many cases of chronic otitis media which did not become dry under the usual types of treatment were allergic in character." In a few cases eosinophiles were found in aural secretion in sufficient numbers to establish a diagnosis of allergy. This finding has not been consistent and has not been considered a reliable diagnostic procedure. More emphasis can be placed on the intermittent, profuse serosanguineous discharge associated with allergic exacerbations. The antihistaminics have proved very valuable as a diagnostic aid in this problem.

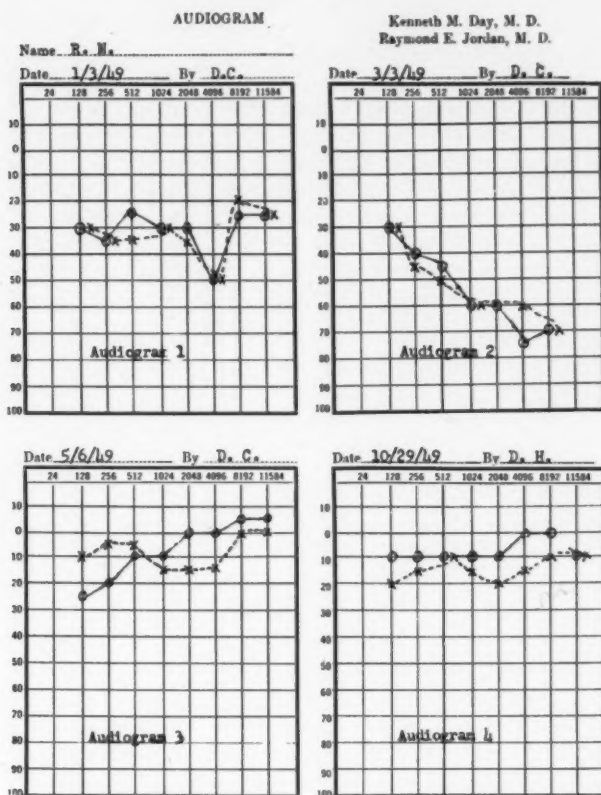


Fig. 3. A case of Ménière's disease without vertigo treated with subcutaneous histamine for 10 months.

Allergy of the inner ear offers a very interesting and speculative field. Much has been written concerning the pathology, signs, symptoms and treatment of Ménière's disease. The results of treatment with histamine as reported by Lillie, Horton and Thorn⁸ and others place the etiology in the allergic category. Williams,¹¹ in 1947, reported 32 cases which he termed endolymphatic hydrops without vertigo, giving the pathophysiology and results of treatment with intravenous

histamine. My review of 110 cases of Ménière's disease with or without vertigo adds nothing new to what has already been written on the subject; however, a different therapeutic approach has been used. No criticism of the results of the intravenous histamine method of treatment for Ménière's disease is intended, but it does have certain limitation because of time and equipment required.

A breakdown of the above mentioned 110 cases is as follows: Eighty-two cases were diagnosed as Ménière's disease with vertigo, while 28 cases were diagnosed as allergic deafness or Ménière's disease without vertigo. The allergic treatment of the 82 cases with vertigo was under the management of Dr. Florence Kline,⁷ of Pittsburgh. The treatment consisted of weekly or biweekly subcutaneous injections of minimal doses of histamine base, and other supplementary allergic therapy as indicated. The results of this treatment were as follows: Vertiginous attacks were controlled in 41 cases. Nineteen cases improved but continued to have some vertigo. Twenty-two cases had insufficient clinical observations to report. The improvement in hearing and the change in tinnitus was insignificant in these cases.

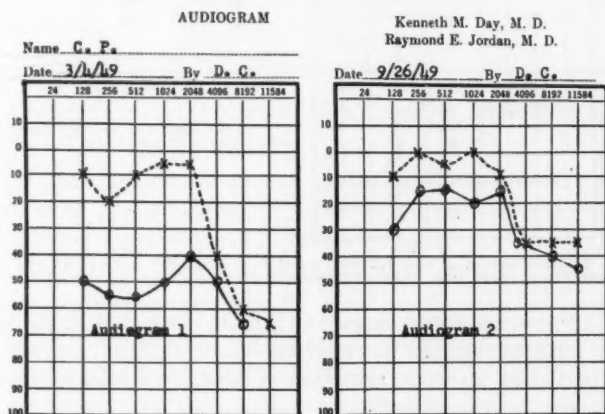


Fig. 4. A case of Ménière's disease without vertigo treated with anti-histaminics and elimination diet.

The controlling dose of subcutaneous histamine in 80 per cent of the 41 cases in which the vertigo was controlled varied between 0.2 and 0.5 cc. of 1:100,000 dilution of histamine base.

Of the 28 cases of Ménière's disease without vertigo, 15 were treated by a similar method, with the addition of antihistaminics in some cases. The results of treatment were as follows: Seven cases recovered hearing to the conversational level, four improved but did not reach the conversational level, and four were not improved audiometrically. An illustration of a case treated with subcutaneous histamine for 10 months is shown in Fig. 3. Fig. 4 demonstrates the hearing changes in an early case of Ménière's disease without vertigo which occurred after treatment with antihistaminics and elimination diet.

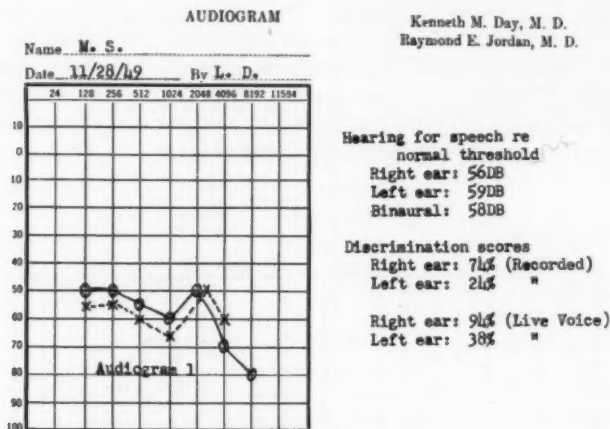


Fig. 5. Illustration of the difference between the pure-tone level and the discrimination score in a case of Ménière's disease.

The following three cases are illustrations of allergic tinnitus without vertigo or hearing change. This finding is interpreted as an early symptom of Ménière's disease.

Mrs. W. W., age 27, a physician's wife, was first seen in April, 1949, with a history of high-pitched tinnitus in the left ear for two and one-half years. Two weeks prior to the patient's first visit, the tinnitus had

increased in loudness and was constant. The physical examination was essentially negative. The nasal smears were positive for allergy. Skin tests revealed a sensitivity to dust, milk and wheat. These foods were eliminated from the diet and thephorin,¹⁰ 25 mg., was prescribed three times a day after meals. Two months later the patient was free of symptoms. For the past year the patient has not had tinnitus except when thephorin is discontinued.

Mrs. R. A., age 59, whose chief complaint was a hissing tinnitus in both ears, was a similar case. Tagathen,⁹ when taken, eliminated the tinnitus.

A third case, Mrs. J. M., age 59, complained of a low-pitched roar for 10 weeks. Sensitivities to orange, egg and chocolate were found, and improvement occurred with elimination of these foods. The patient discovered that with the elimination of grapefruit the tinnitus disappeared completely and has remained absent for six months except when grapefruit is eaten.

It was not possible to draw any definite conclusions in the study of these cases concerning the relationship between the duration of symptoms, the amount of hearing loss and the results of treatment; however, if treatment was not instituted prior to the onset of vertigo, the results in regard to hearing improvement and the loss of tinnitus were poor. It has been noted in many of these cases that the discrimination did not approximate the pure-tone level. Because of distortion, a mild loss of hearing for pure tones may be a severe loss for speech. An example which shows an unusual difference is shown in Fig. 5. Further studies of this unusual phenomenon are being carried out.

CONCLUSIONS.

Allergy plays an important rôle, either as a cause or a contributing factor, in deafness.

Clinical evidence indicates that allergy of the middle ear does exist and accounts for a higher percentage of the cases of chronic secretory otitis media.

Perforation of Shrapnell's membrane can be a sequela of chronic secretory otitis media.

The results in fenestration and mastoid surgery are complicated by nasopharyngeal allergy.

Some form of allergy does affect the inner ear. Deafness and tinnitus may occur without vertigo in Ménière's disease.

Early diagnosis and treatment result in restoration of hearing in some cases.

BIBLIOGRAPHY.

1. ASHLEY, REA E.: Medical Care and Prophylaxis in Hearing Losses with Special Attention to Allergies. *Trans. Amer. Otol. Soc.*, 37:169-177, 1949.
2. DAY, K. M.: Primary Pseudocholesteatoma of the Ear. *Arch. Otolaryngol.*, 34:1144-1151, 1941.
3. DEAN, L. W.; AGAR, J. S., and LINTON, L. D.: Allergic Diseases of the Ear. *THE LARYNGOSCOPE*, 47:707-728, 1937.
4. DOHLMAN, F. G.: Allergiska Processer I Mellanorat. *Nord. Med. tidskr.*, 20:2231, 1943.
5. JONES, M. F.: Manifestations of Allergy in the Ear. *Ann. Otol., Rhinol. and Laryngol.*, 47:910-916, 1938.
6. KOCH, HJALMAR: Allergical Investigations of Chronic Otitis. *Acta Otolaryngol.*, Suppl. LXII, 1946-1947.
7. KLINE, FLORENCE, Allergist, University of Pittsburgh: Personal communication.
8. LILLIE, H. J.; HORTON, B. T., and THORNELL, W. C.: Ménière's Symptom Complex: Observations on the Hearing of Patients Treated with Histamine. *Ann. Otol., Rhinol. and Laryngol.*, 53:717-741, 1944.
9. Tagathen, 25 mg., Antihistaminic. Lederle.
10. Thephorin, 25 mg., Antihistaminic. Hoffman-La Roche.
11. WILLIAMS, H. L.; HORTON, B. T., and DAY, L. A.: Endolymphatic Hydrops Without Vertigo. *Trans. Amer. Otol. Soc.*, 35:116-140, 1947.

STANDARDIZATION OF PURE TONE AUDIOMETER TESTING TECHNIQUE.*

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This paper presents: I—an enumeration of some of the factors which influence the accuracy of measurement of auditory pure tone threshold acuity; II—a brief discussion of the application of psychophysical methods to the determination of sensory thresholds; III—a review of the testing procedures usually employed in clinical pure tone audiometry; IV—an evaluation of audiometry as art or science; V—a description of a constant reproducible pure tone presentation technique which also indicates the mathematical probability of the accuracy of patient response; VI—a brief evaluation of two other types of audiometers (previously described in the literature) with which uniformity of tone presentation and highly objective analysis of patient response are attainable.

— I —

Among the factors which influence the reliability of pure tone audiometer test results are the following: 1. the accuracy of the calibration of the audiometer; 2. the magnitude of the masking effect of ambient noise in the testing environment, which includes noise produced by the audiometrist, the patient, and any sound in the receiver resulting from operation of the audiometer other than the desired test tone; 3. position and pressure (coupling) of the sound reproducer in relation to the auricle or bones of the head; 4. physical conditions within the test room which influence body comfort, such as temperature, humidity, barometric pressure, altitude,

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and time of day; 5. age, intelligence, reaction time and previous test experience of the patient; 6. physiologic condition and mental attitude of the patient (alert, interested, relaxed, cooperative, indifferent, lethargic, depressed, fatigued, tense, apprehensive or antagonistic). Patients who experience tinnitus or auditory after-images, malingerers and those with hysterical perceptual abnormalities present additional test difficulties; 7. training experience, insight and personality of the audiometrist; and 8. the complexity of the stimulus presentation technique used in the determination of the auditory threshold. Ambiguity of response instructions to the patient and the use of complex recording methods may be responsible for occasional errors in the patient's response or in the recording of the test data.

Westlake¹⁸ has reported that the heavy "normal" line on the audiogram blank also is responsible for a common source of error. He found that if audiometrists give little or no attention to the decibel readings of the attenuation dial in relation to the zero reference level while testing, the results tend to vary from the "normal" line somewhat more than if they attend closely to the dial readings during the test. Brogden and Miller³ have defined a "Physiological Noise Generated Under Earphone Cushions" as high as 25 to 30 db above the normal threshold value in earphones with small cavities, resulting from tremors of the muscles of hands and arms while holding the phones to the ears. This type of noise, of course, is minimized, if not eliminated, by the use of a well designed headband to hold the phones firmly in place against the ears. It is apparent that most of the above factors which influence the accuracy of measurement of auditory thresholds may be classified under the following three subdivisions: 1. physical considerations; 2. variables associated with the patient; and 3. variables introduced by the audiometrist.

It is highly desirable that every audiometrist possess at least an elementary appreciation of the meaning of the term *threshold* or *limen*. Woodworth¹⁹ states: "... It is a fundamental fact of psychology, and of physiology as well, that,

at any one moment, a stimulus must reach a certain strength in order to be perceived or to elicit a response. . . . As its name implies, the threshold is a *transition point*. . . . The stimulus threshold is the transition point between stimuli too weak to elicit a response and stimuli strong enough to elicit that response."

Guilford⁸ writes: ". . . There is no *single* stimulus value at any point on the scale below which a response never occurs and above which a response always occurs. There is a rather indefinite, twilight region of uncertainty, in which part of the time a response occurs and part of the time it does not. It is generally agreed that we should adopt that stimulus value which gives a response exactly half the time; in other words, the stimulus that has a probability of 0.5 of producing a response, as the *stimulus limen* RL. Above the RL, stimuli give a response more than half the time; below it they elicit a response less than half the time. . . ." Thurstone¹⁷ points out that "The subject varies from moment to moment in his sensitivity and attention, so that it usually becomes necessary to define the limen as a statistical measure."

The absolute threshold of audibility of the "normal" ear usually is expressed in terms of the magnitude of the minimum sound pressure in watts or dynes per sq. cm. at a given point and frequency level required to elicit a response one-half the number of times it is presented. As is well known, clinical audiometers enable threshold measurements to be obtained in terms of decibels above or below the "normal" hearing zero reference intensity level, usually in multiples of 5 db intensity step intervals.

Myers and Harris¹¹ differentiate between ". . . a threshold of detectability and a so-called pure-tone threshold." Their data ". . . indicates that a zone of detectability of the order of 2 to 3 db exists between the intensity at which a tone is just detected 50 per cent of the time and the intensity at which it may be assigned a pitch quality of 50 per cent of the time." In a later article Myers and Harris¹² define this zone

of detectability as follows: "This zone of detectability is defined as the intensity area between a 50 per cent detection threshold and 50 per cent pure-tone threshold." Pollack¹³ also makes a distinction between "thresholds for the detection of the presence of a sinusoidal signal (thresholds of audibility and thresholds of tonality). . . . At each of the frequencies tested, the threshold of audibility was found to be significantly lower than the threshold of tonality. The difference between the two thresholds — the atonal interval — ranged from about 2.5 db at 500 cps. to about 6.5 db at 4,000 cps. Little difference was found between the variabilities of the two thresholds."

These studies by Myers and Harris, and Pollack indicate the advisability of differentiating between the detectability and pure tone thresholds while obtaining auditory acuity measurements, especially for research purposes. The observers must appreciate and experience the difference between the two types of auditory thresholds in a practice session and understand the exact nature of the stimulus to which they are to respond if pure tone threshold acuity data of the maximum possible accuracy are desired.

— II —

According to Chapter 12 of Garrett⁷ since about 1829 considerable literature has developed around the subject of psychophysics, the branch of psychology devoted to the study of perceptual and sensory phenomena, with emphasis on the methods of determining sensory acuity and efficiency.

The psychophysical technique most suitable for the measurement of threshold acuity is the *method of limits*, also called the method of minimal changes or the method of serial exploration. The technique of the method of limits may be applied in the determination of auditory threshold values as follows: By means of a physically measurable and controllable sound source present the test frequency at a definitely audible intensity level and progressively decrease the intensity in

small* intervals until the sound can no longer be heard—a descending series. Then reverse this procedure and increase the intensity until the tone can be heard—an ascending series. In theory, the transition point or threshold stimulus value lies between the last audible and the first inaudible presentation of the descending series or the last inaudible and first audible presentation of the ascending series; however, it has been found that the transition point in the descending series usually occurs at a slightly lower intensity level than the transition point in the ascending series. Consequently it is customary to regard the mean value of the last intensity level on the descending series and the first intensity level on the ascending series at which the observer O heard the tone as the threshold stimulus value. It is advisable to obtain response measurements on several pairs of descending and ascending stimuli for each O at each test frequency in order to obtain sufficient data for adequate statistical evaluation of the accuracy of the threshold measurements.

In order to minimize the errors of habituation and expectation inherent in the descending and ascending presentation of stimuli, it may be expedient to avoid the systematic progressive descending and ascending approach and present the stimuli in a haphazard or chance order. This procedure has been called the method *without knowledge*. For a detailed description of the psychophysical methods and their statistical analysis, see Guilford,⁸ Woodsworth,¹⁹ Thurstone¹⁷ and Chapter 12 of Garrett.⁷

— III —

A review of the relatively few descriptions of audiometer testing technique in the literature suggests that audiometrists

*For the maximum attainable accuracy of measurement the discrete intensity step intervals should be slightly less than the intensity difference limen DL of the ear. The DL may be defined as the stimulus difference that can be perceived 50 per cent of the time. The sound intensity DL is a complex variable being a function of both the frequency and intensity levels at which measured. Riesz,¹⁵ using a method of beats and 12 observers, found that at a sensation level of 5 db, the unilateral differential sensitivity varied from 2.48 db at 4,000 cycles to 9.32 db at 35 cycles. These data suggest that an intensity step interval of the order of 2 db is satisfactory for use in obtaining threshold data, although step intervals of 1 db or even less may be advisable in certain types of threshold measurements.

generally use an exceedingly abbreviated and modified form of the psychophysical method of limits for the clinical testing of auditory acuity. It may safely be assumed that these modifications are subject to considerable (if not infinite) variation in practice, since there seems to be no one generally accepted or standardized testing method. Consequently there are as many variations in exact procedure as there are audiometrists. Undoubtedly these variations in testing technique contribute in part to the well known observation that various audiometrists occasionally obtain significantly different results (audiograms) on a given patient with the same audiometer.

There appears to be ample justification for more or less modification of the classical psychophysical method for the clinical testing of auditory acuity. The psychophysical approach stresses the highest possible attainable accuracy under carefully controlled laboratory conditions. This methodology necessitates that the testing be done in a noise-free environment in terms of intensity increments that approximate a just noticeable difference (j.n.d.), and that a sufficient number of observations (responses) be obtained at each frequency level to amass sufficient data for significant statistical evaluation. The length of time required for the measurements and the statistical computation usually is of secondary consideration since the major emphasis is placed on accuracy of results.

In the average otologic clinic or office, however, a premium is placed on the minimum amount of time required to obtain measurements that are sufficiently accurate to indicate significant diagnostic information. The audiometer air conduction acuity measurements also may be augmented with bone conduction tests, some type of speech test, a test for recruitment and a test to indicate the presence or absence of stapes fixation, all of which are evaluated in relation to the history and physical examination of the ears, nose and throat of the patient. Under these circumstances the clinical audiometrist has been compelled to develop a technique by means of which significant hearing impairments could be detected with relatively little expenditure of time.

In the absence of a standardized test procedure it is not surprising that differences exist among the published descriptions of audiometer testing techniques. Bunch⁴ recommends the following stimulus presentation technique (after instructing the patient to press a noiseless key every time he hears the test tone): "The sound should at first be loud enough for him to hear without any doubt. He should be told that the sound will be turned off and on and will become weaker and weaker until it can no longer be heard. As soon as these instructions are understood, the tone is presented through the receiver and the listener presses the key. The examiner interrupts the sound immediately, decreases the intensity and presents the decreased stimulus to the listener who responds as before. The process is repeated, the stimulus sound being made fainter each time until the listener no longer responds. After a few trials, a sort of rhythm can be set up. The stimulus is presented, the patient responds, the operator interrupts the sound, decreases its intensity and presents it again to the listener. This is repeated until the thresholds for all tones are determined."

This quotation indicates that Bunch favors a procedure in which the intensity is decreased systematically from audibility to inaudibility. Hughson and Westlake⁹ recommend a presentation technique which emphasizes the increase of the intensity from inaudibility to audibility. "A reasonably loud signal should be presented first, basing the intensity on the apparent hearing loss. Attenuation is then introduced in 5 or 10 db steps until no response is indicated. Then take out attenuation until the tone is heard. This maneuver should be repeated and a uniform response obtained at least three times for each frequency involved. The interrupter button should be used whenever the attenuator dial is changed and the length of time the tone signal is presented should be constantly changed, thus avoiding any possibility of rhythmic response."

The writer dares add still another (and probably superfluous) description of pure tone audiometer tone presentation technique to the literature. The only justification for doing so is to present a detailed step-by-step procedure for begin-

ning audiometry students to follow until they have acquired sufficient practice and experience to make modifications on the basis of their own experience. A basic understanding of the factors involved in accurate testing necessitates that certain threshold-seeking maneuvers be employed, which are used in common by all effective audiometrists.

During the testing procedure, it is advisable to rotate the attenuation dial only while the tone is suppressed by means of the tone interruption switch, if rotation of the dial results in perceptible contact noise or "static" in the receiver which cannot be eliminated by cleaning the contacts of the attenuator with carbon tetrachloride and lubricating them with a thin film of a high grade light oil. It is assumed that the audiometrist will know the intensity level settings of the attenuator at which contact noise stimulates the threshold of the normal ear at the different frequency levels generated by his audiometer.

A brief statement to the patient concerning the purpose of the test and an explanation of how he is to cooperate during the test constitute an important part of the procedure. The patient may be "briefed" as follows: "I want to find out how softly you can hear several different tones in each ear. We will test one ear at a time the better ear first, if you think you can hear better with one ear than the other. Each tone will be made comfortably loud at the beginning so you will know what it sounds like and will know what to listen for. Every time you hear the tone, no matter how loud or how soft, push down on this signal key to let me know you are hearing it. Push down on the key as soon as you hear the tone and keep it down as long as you can hear it. When you stop hearing the sound, take your fingers off the key and wait until you hear it once more before you push down on the key again. Since I want to find out how softly you can possibly hear the tone, be sure to push down on the key even when it is so soft you can just barely hear it." These instructions, of course, may be modified to conform to the apparent educational and intelligence levels of the patient. It is advisable that the

patient indicate with a suitable signal the entire time interval *during which* the tone is heard for at least two reasons: 1. to check on the patient's accuracy of response at or near threshold levels by varying the length of the time interval during which the tone is presented, as well as the time interval between successive presentations, and 2. to enable the audiometrist to distinguish between patient responses to the sustained test tones and unintentional attenuator contact noise or other types of spurious intermittent noise.

For clinical testing, the writer believes it inadvisable to instruct the patient to differentiate between the threshold of detectability and the pure tone threshold as defined by Myers and Harris,¹¹ or the thresholds of audibility and of tonality as defined by Pollack.¹³ (It is hoped that a uniform terminology will soon be adopted to designate the "awareness" and "recognition" thresholds defined by these investigators.) Distinctions between these two different sensory aspects of a given tone as its intensity is varied about the threshold, however, cannot be ignored in research studies concerned with highly accurate threshold determinations.

At the beginning of the test make certain that the audiometer attenuation dial is set at or near the zero (normal hearing) reference level before the earphones are placed over the patient's ears. Start the test at a frequency level of 1000 (or 1024) cycles per second. Proceed as follows: *Step 1:* Starting with the intensity level below the patient's threshold, increase the intensity of the tone about 10 db every two seconds by rotating the audiometer attenuation dial in the clockwise direction until the patient indicates that he hears the tone (which may be as high as 10 db or so above his actual threshold by the time the signal is made.) Note the attenuation dial setting or reading at which the patient indicated the sound was heard and present the intensity 10 db above this value for about three seconds so the patient experiences the tone at a comfortable listening and identification level. *Step 2:* Decrease the intensity below this level at a rate of about 5 db every two seconds by rotating the attenua-

tion dial in the counter-clockwise direction until the patient releases the signal key, noting the level at which the key was released. *Step 3:* Increase the intensity above this subliminal level in 5 db steps every three seconds until the patient responds, again noting the level at which the patient indicated the tone was heard. (This second response to the sound usually is within 5 db of the patient's actual threshold value. Note that with three rotational movements of the attenuation dial, a clockwise, a counter-clockwise and a second clockwise rotation — a close approximation to the patient's threshold has been reached.) From this point on, the intensity of the tone is alternately decreased below audibility (as in Step 2) and increased to audibility (as in Step 3) until the patient responds consistently at a given minimum intensity level, 5 db below which there is haphazard or no response.

At least two and preferably three consistent responses at the same minimum level constitute a reasonably accurate indication of the patient's acuity for the frequency in question, assuming synchronous responses to the variations in the time intervals between tone presentations and the duration of the tones, both of which must be varied in successive presentations in manually controlled audiometry.

If the patient appears to respond quickly and accurately at a given level, for example, 45 db, but slowly and somewhat haphazardly at the 40 db level, the audiometrist must adopt a "two out of three" or preferably a "three out of four" synchronized response requirement as the criterion of the correct threshold value for the frequency in question. For example, if the patient responds synchronously to three out of four presentations of the tone at the 45 db level but entirely haphazardly at the 40 db level, then the acuity would be recorded at the 45 db level.

If the patient's responses become increasingly erratic at near threshold levels, it is advisable to present the tone again at a level about 10 db above his apparent or approximate threshold to refresh his memory of the attributes of the tone for which he is listening.

It will be noted that the above approach to the threshold emphasizes the ascending or increasing intensity technique in which the intensity is gradually increased from inaudibility to audibility. A brief review of the entire procedure shows that the tone is presented alternately above and below the patient's threshold, progressing gradually from levels definitely above threshold to the threshold level. In Step 1, the tone is first presented at a comfortable listening level, in Step 2 is attenuated below audibility and in Step 3 is increased to five or possibly 70 db above the threshold. In succeeding steps the tone is alternately attenuated below audibility and increased in intensity in 5 db increments until a reasonably consistent response is obtained at a stable minimum intensity level.

Fortunately less time is required to obtain a threshold measurement as outlined above than is expended in reading the description of it. As indicated previously, the above procedure is merely one way of ascertaining the acuity for a given frequency. Undoubtedly threshold measurements of equal or possibly higher accuracy can be obtained with radical modification of the suggested technique. It is intended that the described procedure serve merely as a detailed guide for the beginning student of audiometry until he finds it expedient to make modifications on the basis of his accumulated experience. Needless to say, considerable modification of technique may be required to obtain reliable acuity measurements on young children, senile individuals and patients with hysterical or other types of perceptual abnormalities.

It is merely a matter of time until one or more of several nationally recognized professional groups or committees interested in the accuracy and reliability of audiometric measurements will recommend that certain aspects of manual audiometry be standardized. For example, general agreement on whether the auditory acuity should be ascertained by means of an ascending or descending intensity series (or a combination of both), the number of correct responses that should be required to indicate the threshold out of a given number of stimuli presentations at near the threshold level, and pos-

sibly the order in which the various test frequencies should be presented are questions about which there should be no uncertainty. Certainly the more uniform the test procedure, the less the confusion in the teaching and the practice of audiometry, with a resultant increase in the accuracy and reliability of test results.

— IV —

Unfortunately the belief is all too prevalent that since the audiometer is a so-called "precision" instrument designed for the scientific measurement of auditory acuity, the results of hearing tests made with the audiometer are correspondingly precise and accurate. Nothing could be further from the truth. Few will deny that most of the functional and operational features embodied in the modern clinical audiometer are based on considerable research and engineering skill; however, present day use of the audiometer leaves much to be desired from the scientific point of view. One of the most fundamental and elementary tenets of science is the application of an exact measurement technique under carefully controlled conditions. The "measuring stick" itself must remain an invariable factor and must be applied and evaluated in precisely the same way each time it is used. Its limits of accuracy (error of measurement) also must be known.

Due to the fact that the presentation of the test tones (the measuring stick) of the usual clinical audiometer is under the manual control of the audiometrist, there are as many variations in testing procedure (as many different ways of applying the yardstick) as there are audiometrists. As indicated previously, no two competent audiometrists could possibly test a given patient in precisely the same way, nor could a given audiometrist test the same patient twice in exactly an identical manner. If similar audiograms are obtained in either instance the skill of the audiometrist is principally responsible — not the naive assumption that since the audiometer is a precision instrument for the accurate testing of auditory acuity, hearing tests made with an audiometer are correspondingly precise. So long as every operational step in the testing

procedure is dependent upon the manual control of the audiometrist, audiometry is and will remain principally an art rather than a science. Consequently an effective testing procedure can be acquired and applied only through practice. No one can make accurate hearing tests with the usual clinical audiometer until he is so familiar with the mechanical operation of the instrument and an adequate tone presentation sequence that his attention is focused on the patient's response rather than the manipulation of the instrument's controls during the testing procedure.

A second factor which supports the viewpoint that present day audiometry is more an art than a science is the fact that the evaluation of the patient's responses often is a matter of subjective judgment, especially when the patient makes frequent incorrect or haphazard responses during the testing procedure. When the response of such a "difficult" patient is synchronized with a tone presentation the audiometrist often wishes he knew if the response were merely chance coincidence or a significant response. If patients responded only during the presentation of tones and never made haphazard judgments, audiometry would be very simple indeed. It is a certainty that Bunch⁴ was concerned less with the difficulties inherent in the mechanics of the operation of the audiometer than with the problem of the evaluation of the patient's responses when he wrote: "The reliability of an audiogram is directly proportional to the skill of the examiner."

The writer does not wish to foster the impression that the art of audiometry is inherently a grossly inaccurate and unreliable test procedure. A certain amount of skill necessarily always will be required to manipulate the controls of equipment which require frequent adjustment or attention from the operator. Obviously any decrease in the amount of manual control needed for the effective operation of a given instrument will result in more uniform and exact performance of that instrument. Audiometry will more nearly approach the scientific goal of measurement in proportion to the degree that the tone presentation technique attains invariable uni-

formity and the evaluation of the patient's responses becomes subject to quantitative verification.

— V —

It is obvious that a type of audiometer which would facilitate the production of test tones in a predetermined reproducible sequence or pattern, which did not present clues that would enable the patient to make correct responses when the tone was presented but not heard, and which permitted an objective quantitative evaluation of the patient's responses would possess certain definite advantages. Among these advantages are the following: 1. Any given patient could be retested any desired number of times with an identical tone presentation technique; 2. any desired number of individuals could be tested with the same invariable reproducible tone presentation sequence; 3. if sufficiently simple to operate mechanically, an individual with very little training in audiometry could obtain results as reliable as an expert audiometrist and different audiometrists would obtain more nearly the same results on the same patient; and, 4. if generally adopted, such an instrument would make possible the standardization of audiometer technique with increased universal accuracy of test results. Such an instrument is highly desirable from the research point of view. And to the extent that the tone presentation technique attained invariable reproducibility and the evaluation of the patient's responses approached objectivity, the testing of auditory acuity with such an instrument would be less an art and a skill and become a more scientific procedure.

An instrument constructed by the writer in 1944 meets the criteria suggested above in a reasonably satisfactory manner. As is often the case, since building this instrument, Gardner² pointed out that practically an identical design was suggested by Munson in a Bell Telephone Laboratories memorandum, which to my knowledge was not published in accessible literature until 1947. The first description of the mechanical tone interrupter devised by the writer is contained in a Master of Arts thesis by Bartell,¹ placed in the State University of Iowa

Medical School Library in August, 1945. Bartell¹ states: "The purpose of this thesis, then, is twofold: first, to describe a technique by means of which individuals can be tested and retested in an exactly reproducible manner, and second, to investigate the variability of normal bone conduction measurements in comparison to normal air conduction measurements. . . . The major apparatus consisted of an audiometer and an automatic stimulus interruption device."

The instrument consists essentially of a motor driven tone interruption device which can be attached to a commercial audiometer. Functionally, the gadget is used to produce one of four stimulus patterns as one, two, three or four dashes or tone pulses coincident with the presentation of an attention light. A quiet fractional horsepower motor is geared to rotate a shaft 6 inches long and $\frac{5}{16}$ inch in diameter through one revolution every 10 seconds. Five bakelite discs, each $\frac{3}{16}$ inch thick and 3 inches in diameter, are mounted rigidly on the shaft. A small leaf-spring switch is mounted near the periphery of each disc so that teeth-like projections, or the depressions between them, activate the switches to interrupt the test tones from the audiometer and turn on a signal light to indicate the interval during which the test tone will be presented as one, two, three or four pulses. The signal light disc (Disc No. 5) is cut with a $\frac{1}{8}$ inch projection or tooth extending $\frac{6}{10}$ of the periphery of its circumference (216 degrees). This projection closes a switch which turns on the signal light for a time interval of six seconds. The light, of course, is off the following four seconds. During the four second interval the light is off, the patient indicates the number of tone pulses heard and the audiometrist selects the desired frequency, intensity and pulse pattern which he desires to present next.

Disc No. 1 is cut with a depression $\frac{1}{8}$ inch deep extending around $\frac{1}{10}$ of its periphery (36 degrees) which presents the tone $\frac{8}{10}$ of a second during a revolution of the disc. Disc No. 2 has two depressions of the same dimensions as that on Disc No. 1, separated $\frac{1}{20}$ the circumference of the disc (18 degrees from each other). As Disc No. 2 revolves, the tone

is presented as two pulses or dashes, each pulse being $\frac{8}{10}$ of a second in duration, separated by a silent interval of one-half second. Discs 3 and 4 present three and four pulses, respectively, the dimensions of the projections and spaces between them being constructed to conform to the dimensions described for Discs 1 and 2. Each of the four pulse-patterns is phased on the shaft in relation to the signal light disc so that the signal light is always illuminated 0.4 second before a pulse is presented. The light is extinguished 0.1 second after the termination of the last pulse produced by Disc No. 4. A selector switch permits selection of the desired pulse pattern for each rotation of the rotor assembly.

Attachment of the mechanical tone interruptor to the audiometer does not disturb the frequency and intensity calibration of the instrument. The leaf-spring switches associated with the pulse pattern discs are connected in parallel with the tone interruption switch of the audiometer, which does not interfere with the manual operation of the audiometer tone interrupter switch; however, it may be necessary to modify certain circuit constants in the interrupter switch circuit of the audiometer to obtain optimum "attack" and "release" times at the initiation and termination of the pulses which are free from transient "clicks." A time interval of 0.1 second for both the attack and release of the pulses has been found satisfactory. The minimum duration of the "steady state" presentation of each pulse should exceed 0.2 second, since Munson¹⁰ reports that: "Full loudness is not reached until over 0.2 second has elapsed." As indicated previously, the duration of the pulses of the present interrupter is 0.8 second. Further experimentation with the device is to be undertaken to ascertain the optimum relations between the duration of the pulses and intervening silent intervals in order to learn the minimum time required for reliable pulse pattern audiometry.

Very little more time is required to test the acuity by means of the pulse tone audiometer than is expended in manual audiometry. It may be found advantageous to combine the usual manually controlled audiometer tone presentation and

the mechanically (or electronically) controlled pulse tone techniques in certain clinical applications as follows: first, determine the approximate threshold level according to one of the manual approaches described earlier; then employ the pulse tone presentation technique. The patient is instructed to listen for one, two, three or four tone pulses or dashes each time the light is on and to indicate the number of pulses heard immediately after the light goes off. Needless to say successive pulse patterns must be presented in random order.

The first pulse pattern may be presented 10 or 5 db above the approximate threshold. Each succeeding pattern is attenuated 5 db until either an incorrect or no response is obtained. Then increase the intensity of the next pattern 5 db. If the pulse pattern is heard correctly at this level, present a second pattern at the same level. If the second pattern also is heard correctly, attenuate 5 db and note the response. Determine the lowest intensity level at which at least two out of three patterns are recognized correctly. Due to the fact there are no defined criteria of correctness of response in terms of pulse tone audiometry, the writer, at the present time, arbitrarily regards the correct clinical threshold value as the lowest intensity level at which at least two of three pulse patterns are recognized correctly, assuming two out of two correct responses for the adjacent 5 db higher level.

As previously intimated, the technique of pulse tone audiometry includes an objective evaluation of the mathematical probability of the accuracy of the patient's response. In order to ascertain the probability of a repeated or combined event, the probabilities of the single events are multiplied. Assuming haphazard or chance presentation of any one of four different pulse patterns, the patient's chance of guessing correctly the single inaudible pattern is one in four, of guessing correctly two successive inaudible patterns is one in sixteen, of guessing correctly three successive inaudible patterns, one in sixty-four and so on. Application of the terms of the *binomial expansion* shows that the patient has nine chances in sixty-four (approximately one in eight) of guessing correctly any two

inaudible patterns out of three, and 27 in 64 of guessing correctly any one pattern out of three. (For a discussion of probability statistics, see Guilford* or any standard text of statistical methods.)

Since the patient has an almost 50-50 chance (27 in 64) of guessing correctly one out of three inaudible patterns, the writer believes it is reasonable to require two correct responses out of three pattern presentations, in spite of the fact the patient has only nine chances in sixty-four (approximately one in eight) of guessing correctly any two inaudible patterns out of three inaudible pattern presentations. This requirement may seem excessively cautious in view of the fact that the auditory threshold is defined in terms of the minimum intensity at a given frequency level that has a probability of 0.50 of producing a response; however, as indicated previously, this 0.50 value is ascertained by means of a psychophysical technique which presupposes the presentation of a sufficient number of stimuli to amass sufficient data for statistical computation and analysis — a procedure requiring radical modification for clinical application. Additional clinical use and research with pulse tone audiometry are needed to determine the most effective testing procedures and to evaluate the usefulness of the pulse tone technique.

Pulse tone audiometry is ideally suited for certain research applications. For such use, it may be advisable to construct a table or key to indicate the intensity levels and pulse tone patterns to be presented at each frequency level. The observer's responses, recorded on an appropriate blank, constitute a permanent record for subsequent analysis. Various types of stimulus keys—ascending, descending or haphazard intensity series—may be devised to meet the requirements of the experiment. In any given key it is essential that the same number of one, two, three and four pulse patterns be used and that the order of presentation of the patterns be due to chance. Of course, keys with more or less than four different pulse patterns may be found advisable for certain experimental purposes.

One of the most effective uses of pulse tone audiometry is the utilization of this technique in the construction of a group pure tone test. Reger and Newby¹⁴ found that reliable pure tone measurements can be obtained on school children down to and including the fourth grade. The group pure tone test detects the existence of high frequency losses which are not revealed by means of the group phonograph record speech (double digit) test.

Gardner,⁶ in 1947, reported the results of several clinical threshold measurements as made with an electronically generated pulse tone audiometer. He found that "... the pulse-tone test procedure gives threshold results which are somewhat more reproducible than results obtained with the standard test procedure, although the time required to make the former is slightly longer. In terms of the attractiveness of the tests for the operator and for the listener, the pulse-tone test appears to be at an approximate three to one advantage. The normal threshold intensity calibrations of the two tests are in good agreement, so that either test may be used interchangeably with the other. For the very young subject, the pulse-tone test appears to be preferable in the majority of cases and tends to lower the age limit at which threshold measurements can be made. . . ."

— VI —

In addition to the mechanical or electronic pulse tone testing technique, at least two other types of pure tone audiometers have been described in the literature by means of which a uniform testing procedure is attainable. Brief functional descriptions of these instruments will be given at the present time to re-emphasize their desirable features and to bring them to the attention of those who may have missed the original articles.

The audiometer described by Burr and Mortimer⁵ consists essentially of a discrete octave audiofrequency generator, an automatic random stimulus selector and a modified electrically activated typewriter by means of which "the patient's replies

are automatically recorded on a card which forms without transcription part of the permanent record." A loud speaker is used for reproduction of the test frequencies in a free field. The test frequencies (125 to 8000 cycles per second in octave intervals) may be presented either as sustained pure tones or as so-called warble tones. "This is a tonal effect produced by varying the frequency over a small range about the mean frequency, the time of a complete cycle of this variation being one-thirtieth of a second." The audiometrist approximates the patient's threshold manually at the beginning of the test at each frequency level and adjusts the attenuator so that the threshold will lie within the intensity range of the test stimuli which are then presented by the automatic random stimulus selector. A signal light glows each time a test tone or a zero stimulus is presented. A "yes" button is pushed by the patient if the tone is heard, a "no" button if not heard.

The test tone is presented 30 times at each frequency level — five times at each of six different intensity levels — over a range of either 20 or 10 db. A zero stimulus is presented once, making 31 trials for the test at each frequency level. For example, assume that a preliminary manual test of a patient's acuity at a given frequency level indicates an approximate 40 db loss. The attenuation control of the audiometer is then adjusted so that the maximum and minimum intensity range of the 30 tone presentations will cover an intensity range of 50 to 30 db in 4 db steps — five times at a level of 50 db, five times at 46 db, five times at 42 db, five times at 38 db, five times at 34 db and five times at a level of 30 db — in random intensity order. (The tone also may be presented in similar fashion over a 10 db range in 2 db step intervals if desired.)

Each intensity level is correlated with a letter on an electrically activated typewriter key. Each time the "yes" button is pushed, the letter associated with the intensity of the test tone just presented is typed on the patient's record; each time the "no" key is pushed a semicolon is typed on the record. The maximum intensity at each frequency level — in this case 50 db — is assigned to the letter "a," which intensity level is

indicated on the patient's record. Since pushing either the "yes" or the "no" button activates the random stimulus selector, thereby ending the present trial and initiating the next one, the rapidity with which the patient responds with the buttons determines the length of time required for the 31 presentations at each frequency level. According to the authors, this time is usually from four to five minutes; therefore, approximately 30 minutes are required for a test at seven different frequency levels.

The audiometrist analyzes the data typed on the patient's record and constructs a graph which plots the number of "yes" and "no" responses against the intensity level if desired. It is apparent that the testing procedure described by Burr and Mortimer conforms to the psychophysical method of limits in which the different intensity levels are presented in a chance or haphazard series.

The audiometer described by Bekesy² is also a self-recording instrument of the continuously variable frequency type which automatically sweeps through a frequency range from 100 to 10,000 cycles per second over a 15 minute interval. The frequency of the oscillator is varied by means of a motor which is geared to rotate a shaft which is attached to the frequency dial or control of the continuously variable audio-oscillator. Mounted on the shaft is a drum several inches in diameter on which is fixed (clipped) an audiogram blank. The patient is instructed to push down on a key until a sound can just be heard. As soon as the sound is heard the key is to be released until it disappears or can no longer be heard, whereupon the key is to be depressed again until the tone can be heard once more, and so on. Pushing on the key starts a second motor which drives back and forth in either direction the slider or contact arm of an attenuation network which covers an intensity range of 140 db in 2 db steps. Attached to the contact arm of the attenuator is an ink pen which records on the audiogram blank the position of the contact arm — the intensity levels — at which the continuously variable tone is heard. The audiogram blank for use with this instrument is specially constructed so that the intensity ordi-

nates and frequency abscissas are correlated with the corresponding positions of the attenuator contact arm and the frequency dial of the oscillator. The recording or responses of the normal ear on the audiogram blank appears as a zigzag line, the median of which is a straight line which indicates the threshold acuity. After instructing the patient and turning on the equipment, the test requires so little attention from the audiometrist that it may be regarded as a self-administering and self-recording test.

The attention lights used in connection with the Burr and Mortimer and the mechanical and electronic pulse tone audiometers, and the varying frequency of the Bekesy instrument are devices which enable the patient to exert maximum attention during the presentation of the test stimuli. Conventional audiometer technique requires that the patient be listening intently with no relaxation of his attention during the critical part of the testing procedure, since he must not know just when a tone at or near the threshold level will be presented. Also he is attempting to indicate the instant the tone is presented and the instant it is terminated. This is expecting a higher order of attention over a longer period of time than can reasonably be expected. Fluctuation in the patient's attention is responsible for much of his erratic response and variability. If by coincidence he happens to be attending at the instant a near threshold level tone is presented, he may hear it; otherwise he may not. Stevens and Davis¹⁶ make an interesting comment on a well-known psychological observation: "The inherent variability of the observer himself can be easily demonstrated by presenting him with a steady tone at an intensity very near threshold and requiring him to press a button during all the time that he hears the tone. Almost without exception observers press the button intermittently."

SUMMARY.

1. Many factors influence the accuracy of clinical audiometric measurements among which are the differences in tone presentation techniques used by various audiometrists and the difficulties inherent in evaluating the patient's responses.

2. Auditory threshold acuity measurements of the maximum accuracy are obtained by means of psychophysical techniques which necessitate securing a sufficient quantity of data under carefully controlled conditions to enable significant statistical analysis and evaluation of the data.

3. Due to "certain" practical considerations (such as limitations on time, personnel, equipment and testing environment), the testing techniques generally employed in clinical audiometry at best must be regarded as exceedingly abbreviated and modified versions of the psychophysical method of limits.

4. Variations in test results between different audiometrists will be minimized by the general adoption of a uniform tone presentation technique, including emphasis on the approach to the threshold (ascending or descending or mixed intensity series), the number of required synchronized responses to the stimulus at a minimum intensity level 5 db below which there is uncertain or no response, the sequence of presentation of the different test frequencies and possibly the duration of the response the patient is instructed to make to the audible test stimuli.

5. At the present time clinical audiometry is more an art than a science.

6. Although the audiometer is generally regarded as a precision instrument for the clinical testing of auditory acuity, the accuracy of the results obtained with the audiometer are limited principally by factors extraneous to the instrument itself.

7. Pulse tone audiometry introduces a testing procedure which possesses the following advantages: *a.* a method of retesting a given patient any desired number of times with an automatically controlled, exactly reproducible, tone presentation technique; *b.* a means of testing any desired number of individuals in an invariable manner; *c.* a technique by means of which a relatively untrained examiner can obtain results as accurate as those of the skilled audiometrist; *d.* the type

of tone presentation makes possible an objective evaluation of the mathematical probability of the accuracy of the patient's response with relatively few data (responses); *e.* the stimuli are presented while the patient is alerted by a signal light to give maximum attention to the stimulus; *f.* there is little or no ambiguity of patient response; and *g.* pulse tone audiometry presents many features which can be standardized.

8. Pulse tone audiometry makes possible a practical group pure tone hearing test.

9. The audiometers described by Burr and Mortimer⁵ and Bekesy² possess desirable self-administering and self-recording features, and constitute uniform testing procedures.

10. Standardization (general acceptance of uniform technique) of manual audiometry and/or of electromechanically controlled audiometers will introduce more science into the art of audiometry.

BIBLIOGRAPHY.

1. BARTELL, B. R.: Variability of Air Conduction and Bone Conduction Measurements in Individuals with Normal Air Conduction Acuity. M. A. Thesis, State University of Iowa, 1945.
2. BEKESY, G. v.: A New Audiometer. *Acta Oto-Laryngol.*, 35:411-422, 1947.
3. BROGDEN, W. J., and MILLER, G. A.: Physiological Noise Generated Under Earphone Cushions. *Jour. Acoust. Soc. Amer.*, 19:620-623, 1947.
4. BUNCH, C. C.: Clinical Audiometry. St. Louis: C. V. Mosby Co., 1943, 186 pp.
5. BURR, E. G., and MORTIMER, H.: Improvements in Audiometry at the Montreal General Hospital. *Canad. Med. Assn. Jour.*, 40:22-27, 1939.
6. GARDNER, M. B.: A Pulse-Tone Clinical Audiometer. *Jour. Acoust. Soc. Amer.*, 19:592-599, 1947.
7. GARRETT, H. E.: Great Experiments in Psychology. New York: The Century Co., 1930, 337 pp.
8. GUILFORD, J. P.: Psychometric Methods. New York: McGraw-Hill Book Co., Inc., 1936, 566 pp.
9. HUGHSON, W., and WESTLAKE, H. D.: Manual for Program Outline for Rehabilitation of Aural Casualties, Both Military and Civilian. Supp. to *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, 15 pp., 1944.

10. MUNSON, W. A.: The Growth of Auditory Sensation. *Jour. Acoust. Soc. Amer.*, 19:584-591, 1947.
11. MYERS, C. K., and HARRIS, J. D.: Detection Thresholds and Pure Tone Thresholds in Auditory Acuity. U. S. Naval Med. Research Lab., U. S. Naval Submarine Base, New London, Conn., 7 pp., 1948.
12. MYERS, C. K., and HARRIS, J. D.: The Emergence of a Tonal Sensation. U. S. Naval Med. Research Lab., U. S. Naval Submarine Base, New London, Conn., 15 pp., 1948.
13. POLLACK, I.: The Atonal Period. *Jour. Acoust. Soc. Amer.*, 20:146-149, 1948.
14. REGER, S. N., and NEWBY, H. A.: A Group Pure Tone Hearing Test. *Jour. Sp. Disor.*, 12:61-66, 1947.
15. RIESZ, R. R.: Differential Intensity Sensitivity of the Ear for Pure Tones. *Phys. Rev.*, 31:867-875, 1928.
16. STEVENS, S. S., and DAVIS, H.: Hearing—Its Psychology and Physiology. New York: John Wiley and Sons, Inc., 1938, 489 pp.
17. THURSTONE, L. L.: Psychophysical Methods, Chapter 5 of Methods of Psychology, edited by T. G. Andrews. New York: John Wiley and Sons, Inc., 1948, 716 pp.
18. WESTLAKE, H.: The Reality of the Zero Reference Line for Pure Tone Testing. *Jour. Sp. Disor.*, 8:285-288, 1943.
19. WOODWORTH, R. S.: Experimental Psychology. New York: Henry Holt and Co., 1938, 889 pp.

OTOGENIC MENINGITIS DUE TO ESCHERICHIA COLI TREATMENT WITH CHLOROMYCETIN.*†

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Escherichia coli meningitis continues to be a rare disease of interest mainly to the pediatrician since it occurs chiefly in the newborn. Schere,¹ in 1895, reported three cases in a series of 1,500 cases of meningitis in children. Craig,² in 1936, reported *Escherichia coli* as the etiological factor in half his cases of meningitis in children. Barrett, Rammelkamp and Worcester,³ in 1942, were able to collect a total of 108 reported cases of *Escherichia coli* meningitis.

The disease is of interest to the otolaryngologist because otitis media and mastoiditis may be the source of infection leading to meningitis as described by Schere,¹ Vonderabe and Baurichter,⁴ Thompson and Quinn,⁵ and Segiet.⁶

Meningitis due to *Escherichia coli* was usually fatal until 1939, when Thompson and Quinn⁵ in this country first reported the successful treatment of the disease with sulfonamides. Other recoveries following treatment with sulfonamides were reported in the next several years; however, treatment was usually prolonged and sequelae such as hydrocephalus and encephalopathy were not unusual.⁷

The introduction of streptomycin with its antibiotic effect on Gram negative organisms offered a new ray of hope in the treatment of *Escherichia coli* meningitis.

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Alexander,⁸ in 1946, reported the successful use of streptomycin in the treatment of meningitis due to *Escherichia coli*. The patient was a 19-year-old soldier and the original site of infection was in the tibia. Streptomycin was administered intramuscularly for 16 days and intrathecally for 12 days, with an additional dose on the fifteenth day. The initial cell count in the spinal fluid was 507 cells, of which 70 per cent were polymorphonuclear cells. Spinal fluid cultures were positive for *Escherichia coli* on the first and fourth days. There was dramatic improvement in the patient's general condition, but the cell count in the spinal fluid remained about 200 until intrathecal treatment was discontinued.

Shields,⁹ also in 1946, reported the successful treatment of *Escherichia coli* meningitis in a five-week-old infant. The patient was given streptomycin by both intramuscular and intrathecal routes for 10 days. The blood culture and spinal fluid culture both showed a pure growth of *Escherichia coli*. The initial cell count in the spinal fluid was 68 cells. The patient recovered without sequelae. At the time of discharge from the hospital, 19 days after admission, the cell count was 120 cells, mostly lymphocytes. At a check-up three days later, it was 154 cells. The spinal fluid culture became negative for *Escherichia coli* two days after the initial dose of streptomycin.

Lieberman,¹⁰ in 1947, reported a third case of *Escherichia coli* meningitis successfully treated with streptomycin. The patient was a nine-month-old infant and the etiology was unknown. Streptomycin was administered intramuscularly for nine days and intrathecally for four days. The initial cell count in the spinal fluid was 760 cells, of which 90 per cent were polymorphonuclear cells. Further cell counts were unreliable as the spinal fluid was persistently blood tinged. The patient recovered clinically on the fifth hospital day and had no sequelae.

The introduction of chloromycetin furnished the physician with an antibiotic having five times the power of streptomycin to inhibit the growth of *Escherichia coli*.¹¹ When adminis-

tered by mouth, the drug rapidly appears in the blood and urine;¹² it also appears in the spinal fluid in a concentration approximately half that noted in the blood, and would appear to make intrathecal administration unnecessary. Clinically, chloromycetin has shown no toxic effects. It is administered orally, and in comatose patients it may be given by gavage. It may be administered parenterally in a solution in proylene glycol, but such usage is not recommended. Because of its characteristics, chloromycetin was selected in treating the following case of *Escherichia coli* meningitis:

History: S. K., a 25-year-old white male veteran, was admitted to the Baker Veterans Administration Center, July 6, 1949. He complained of a chronically discharging right ear dating back to May, 1944, when he incurred a rupture of his right membrana tympani while in the gun turret of a naval vessel. Following this injury, the ear drained intermittently and the discharge had a foul odor. In addition, he had noted some loss of hearing in the right ear. Treatment, including the recent use of aureomycin ear drops, had failed to clear up the discharge. His general health had otherwise been good.

Physical Examination: There was a marginal perforation of the right membrana tympani in the posterosuperior quadrant and a moderate amount of foul-smelling pus coming from the middle ear. There was slight deviation of the nasal septum to the right and some lymphoid tissue in the nasopharynx. Physical examination was otherwise normal.

Laboratory Examination: Examination of the blood showed: hemoglobin, 13 gm.; red blood cells, 4.3 million; white blood cells, 8,500, with 65 per cent polymorphonuclears. The urinalysis was normal. The Kahn test was negative. X-ray examination of the chest showed no abnormality. X-ray examination of the mastoids showed clouding and sclerosis around the right mastoid antrum. The left mastoid showed no evidence of disease. Audiogram showed a hearing loss of 62 db on the right and 6 db on the left.

Clinical Course: On July 12, 1949, under general anesthesia (sodium pentothal), a right modified radical mastoidectomy was performed. There was moderate destruction in the region of the antrum with granulations and a membrane resembling cholesteatoma present. There was a pathological exposure of dura in the region of the tegmen tympani, but the dura appeared healthy. Culture of the mastoid antrum at the time of operation showed a growth of staphylococcus aureus, Alpha streptococcus and *Escherichia coli* colonies. Following operation the patient was placed on procaine penicillin, 300,000 units daily, as a matter of routine. On the second postoperative day, the patient's temperature was elevated to 103.4° F., and the patient complained of nausea and headache. Examination revealed no nystagmus, no Brudzinski sign and no Kernig sign. The patient was placed on penicillin, 50,000 units every three hours. The wound was dressed and all packing removed from the mastoid cavity. Temperature returned to normal and the patient's symptoms disappeared. On July 18, the patient again complained of headache and temperature rose to 101.6° F., with a corresponding rise in pulse and respirations. Blood examination showed a count of 13,600 white cells, of which 68 per

cent were polymorphonuclears. The following day the rectal temperature was 104.4° F. and the patient seemed lethargic. Examination revealed some stiffness of the neck and the Kernig sign was positive. Spinal tap was performed and pink, opalescent fluid obtained. The pressure was elevated to 600 mm. Laboratory examination of the spinal fluid showed a cell count of 4,500, with 95 per cent polymorphonuclears. There was no sugar present. Total protein was over 200 mg. No organisms were seen, but a culture showed a growth of *Escherichia coli*. The patient was given 500 cc. of whole blood and during the next 24 hours he received 20 gm. of sodium sulfadiazine intravenously. Penicillin was increased to 500,000 units every four hours. On July 20, the temperature was still elevated to 103.4° F., and the spinal fluid cell count showed 23,300 cells, of which 96 per cent were polymorphonuclear leucocytes. Smear of the fluid showed a few Gram negative rods. With this report of Gram negative rods found on smear and the culture from the previous day showing *Escherichia coli*, the patient was started on chloromycetin therapy. An initial dose of 3 gm. was given by mouth and followed by 0.25 gm. every four hours. Sulfadiazine was discontinued because urinalysis on this date showed sulfa crystals and large numbers of red blood cells. Ten thousand units of penicillin were given intrathecally at the time of the second spinal tap.

Twenty-four hours after the start of chloromycetin therapy, the patient was less drowsy; however, the temperature remained elevated and there was still present stiffness of the neck. He was taking fluids well by mouth. Spinal tap at this time showed the total white count to have dropped to 6,400, of which 97 per cent were polymorphonuclears. Sugar was still absent from the spinal fluid. The dosage of chloromycetin was raised to 0.5 gm. every four hours. At this time it was decided to convert the modified radical mastoidectomy into a complete radical to remove any possibility of a focus in the middle ear feeding the meninges. This was accomplished on the following day. At operation a few granulations were found over the exposed dura, but there were little additional abnormal findings in the middle ear. The wound was left wide open and packed loosely with plain gauze. The surgery was performed under intravenous pentothal anesthesia, and 500 cc. of whole blood were given following the operation. Spinal tap on this day showed the total cell count to be 3,700 white cells, of which 95 per cent were polymorphonuclear leucocytes. Culture of the fluid showed *Escherichia coli* to be present still.

Following completion of the radical mastoidectomy, the temperature dropped and spinal tap the next day showed only 950 white cells, of which 91 per cent were polymorphonuclears. Sugar was still absent. No organisms were found on smear and no growth was obtained from culture.

By July 27 the temperature had been down to 99° F. for four days. The patient was greatly improved clinically and the stiffness of the neck had largely disappeared. Penicillin, which had been continued because of the initial laboratory report of streptococcus and staphylococcus organisms in the mastoid, had been discontinued three days previously. The patient was taking nourishment well by mouth. Spinal tap on this date showed a white cell count of four, of which two cells were polymorphonuclear leucocytes. There were no organisms found on smear or culture. Sugar for the first time was present to the amount of 22 mg. Blood examination showed 3.1 million red cells, 6,500 white cells and 10.5 gm.

of hemoglobin. Because of the blood findings and the patient's complaint of weakness, another 500 cc. of whole blood were given. Chloromycetin was continued but the dosage reduced to 0.25 gm. every four hours.

On Aug. 1, a plastic closure of the mastoidectomy wound was performed. Three days later, the patient's condition continuing satisfactory, chloromycetin was discontinued. In all, the patient had received chloromycetin for 15 days without toxic effect. The total dosage had been 33 gm.

By Aug. 19 the patient had gained six pounds in weight and the mastoid cavity was becoming covered with epithelium. The patient was given a 10-day leave. Audiogram at this time showed a decibel loss of 46 in the operated ear. This was a gain of 16 db in hearing acuity. At the termination of the leave period the patient was again examined. The radical cavity had become almost completely epithelialized, the hearing gain was maintained and there were no residual neurological findings. The patient was then discharged from the hospital.

COMMENT.

The course of events in this case would seem to indicate a spread of the infection from the mastoid cavity directly to the meninges. The culture taken at operation showed *Escherichia coli* in addition to *staphylococcus aureus* and *Alpha streptococcus*. The rise in temperature two days postoperatively probably heralded a thrombophlebitis of a dural vessel and the second elevation four days later, the beginning of the diffuse meningitis. Blood culture was not taken.

The fact that the meningitis developed while the patient was on penicillin therapy and the failure of the patient to respond to large doses of penicillin would eliminate this antibiotic as a factor in the cure of the *Escherichia coli* meningitis.

The paucity of additional findings at the time the modified radical cavity was converted into a full radical cavity seems to relegate the surgical procedure to a very minor rôle, if any, in clearing up the meningitis.

Under treatment with chloromycetin, the patient's condition quickly improved and the spinal fluid culture became negative for the *Escherichia coli* organism within 72 hours. The cell count fell from 23,300 to 950 in this same period. Since the patient showed no toxic effects from the chloromycetin, it is likely that larger doses might have been used in the beginning

of therapy. The patient was kept on the antibiotic after the spinal fluid became normal because of the necessity of doing a plastic closure of the mastoidectomy wound. It was felt that the surgery might "stir things up," so the chloromycetin was continued until this was completed.

No determinations of the concentration of chloromycetin in the blood or spinal fluid were made.

SUMMARY.

A case of fulminating meningitis due to *Escherichia coli* in a 25-year-old veteran is presented. The clinical course and operative findings indicated the infection to be otogenic. The meningitis rapidly cleared under treatment with chloromycetin by mouth. Although chloromycetin was administered over a period of 15 days, there were no toxic effects. There were no sequelae.

TABLE I. EXAMINATION OF THE SPINAL FLUID.

Date	Cell Count	Polymorpho-nuclears	Sugar	Culture
July 19	4,650	95%	0	<i>Escherichia coli</i>
July 20*	23,300	96%	0	<i>Escherichia coli</i>
July 21	6,400	97%	9	<i>Escherichia coli</i>
July 22	3,700	95%	0	<i>Escherichia coli</i>
July 23	950	91%	0	No growth
July 27	4	50%	22 mg.	No growth

*Chloromycetin therapy started.

BIBLIOGRAPHY.

1. SCHERE, F.: Ein Beitrag zur Aetiologie der Leptomeningitis bei Säugling. *Jahrb. f. Kinderh.*, 39:1, 1895.
2. CRAIG, W. S.: Meningitis in the Newborn. *Arch. Dis. Child.*, 11:171, 1936.
3. BARRETT, G. S.; RAMMELKAMP, C. H., and WORCESTER, J.: Meningitis Due to *Escherichia Coli*; Report of Two Cases with Recovery Following Chemotherapy. *Amer. Jour. Dis. Childhood*, 63:41, Jan., 1942.
4. VONDERABE, A. R., and BAURICHTER, F. X.: *Bacillus Coli* Meningitis. *Ohio State Med. Jour.*, 27:370, 1931.

5. THOMPSON, H. E., and QUINN, R. P.: Meningitis Caused by Bacillus Coll. *Jour. Iowa Med. Soc.*, 29:611, Dec., 1939.
6. SEIGET, W.: Pathogenesis of So-Called Coll Meningitis of the New-born. *Frankfort Ztschr. f. Path.*, 52:208, 1939.
7. STRONG, P. S., and EDWARDS, J. E.: Escherichia Coll Meningitis. *Jour. A. M. A.*, 129:210, Sept., 1945.
8. ALEXANDER, A. V.: Escherichia Coll Meningitis Treated with Streptomycin. *Jour. A. M. A.*, 131:663, June, 1946.
9. SHIELDS, W. P.: Meningitis Due to Escherichia Coll. *Jour. A. M. A.*, 132:514, Nov., 1946.
10. LIEBERMAN, H.: Escherichia Coll Meningitis Treated with Streptomycin. *N. Y. State Jour. Med.*, 47:2206, Oct., 1947.
11. SMITH, R. M.; JOSLYN, D. A.; GRUHZIT, O. M.; McLEAN, W., JR.; PENNER, M. A., and EHRLICH, J.: Chloromycetin: Biological Studies. *Jour. Bact.*, 55:425, Mar., 1948.
12. LEY, H. L., JR.; SMADEL, V. E., and CROCKER, T. T.: Administration of Chloromycetin to Normal Human Subjects. *Proc. Soc. Exp. Biol. and Med.*, 68:9, May, 1948.

**NEUROFIBROMATOSIS (VON RECKLINGHAUSEN'S
DISEASE) WITH INVOLVEMENT OF THE LARYNX.
REPORT OF A CASE.***

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Chicago, Ill.

Although neurofibromatosis (Von Recklinghausen's disease) is a relatively common tumor, it rarely involves the larynx. Review of the literature reveals seven cases previously reported. Neurofibromas of the larynx are benign and cause difficulty only when they invade a vital structure. In the case herein reported, symptoms of the disease began in early childhood but caused no untoward symptoms until the growth in the larynx became large enough to interfere with swallowing and respiration.

CASE REPORT.

J. W., a 27-year-old white male, was seen in the otolaryngologic clinic of the University of Illinois Research and Educational Hospitals in May, 1949. His chief complaint was intermittent dyspnea and dysphagia which lasted for six months. According to the history, tumors appeared on his body when he was a year old and increased in number and distribution to involve the entire body. One such nodule, removed when the patient was eight years old, on histologic examination proved to be sarcoma.

In the Spring of 1948 the patient developed a cough and difficulty in breathing; later, alarming choking spells occurred together with some dysphagia. Examination of the larynx elsewhere had revealed a mass in the larynx, and he was referred for treatment to the University of Illinois Research and Educational Hospitals.

Indirect laryngoscopy revealed a round, pedunculated yellowish mass about 2 cm. in diameter that seemed to arise from the left pyriform fossa. It moved back and forth in the glottis upon respiration.

General physical examination was negative except for numerous nodules over the patient's body and the laryngeal findings described above.

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Direct laryngoscopy confirmed the findings shown by mirror examination. On palpation with forceps the mass was found to be rather firm. The configuration and motility of the cords were normal. An attempt was made to aspirate fluid from the tumor, but no fluid could be obtained, confirming the impression that the mass was not cystic. Tissue was then taken for biopsy and proved to be edematous mucosa, tissue consistent with a polyp.

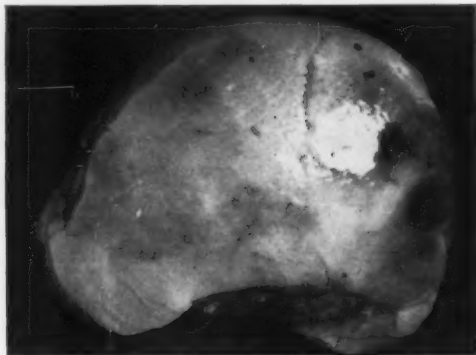


Fig. 1. Gross specimen of neurofibroma of the larynx measuring 3 x 2 x 1 cm.

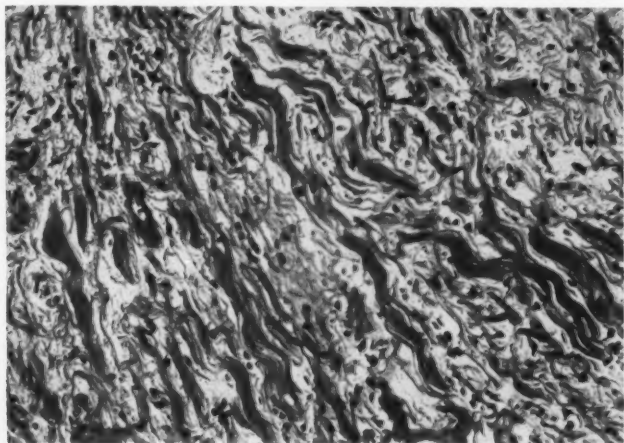


Fig. 2. Photomicrograph showing the characteristic appearance of neurofibroma ($\times 430$).

In the interim, one of the nodules removed from the patient's back proved to be a neurofibroma. Because of this, the mass in the larynx was suspected to be a similar tumor. A second direct laryngoscopy was performed several days later, and the mass was removed by a snare and laryngeal forceps with some difficulty, because of the firm fibrous nature of the lesion. Gross examination showed the tumor to be covered by normal appearing mucosa. Free bleeding ensued from the left pyriform fossa; this subsided rapidly, and the patient returned to the ward in good condition. Several hours later he coughed up bright red blood coming from the site from which the tumor had been removed. Unsuccessful attempts were made to control the hemorrhage by pressure under indirect as well as direct laryngoscopic guidance. Tracheotomy was performed; this allowed for more thorough packing of the pharynx and pyriform sinus, and the bleeding ceased. One thousand cc. of whole blood was administered and the patient was kept on intravenous feedings, strict bed and voice rest, and steam inhalation for a period of five days. The tracheotomy tube was removed on the seventh day, and the patient left the hospital in good condition. Histologic examination of the involved tissue established neurofibroma of Von Recklinghausen's type. Subsequent follow-up examinations have shown the larynx and pharynx to be normal.

DISCUSSION.

Von Recklinghausen's disease (multiple neurofibromatosis) is a relatively common condition,¹ but neurofibroma of the larynx is quite rare. New and Erich² report but one neurofibroma of the larynx seen during a 30-year period. Van Loon and Diamond³ reviewed the literature and found descriptions of only six cases, to which they added one of their own. In only one of the seven previously reported cases of neurofibroma of the larynx was the laryngeal lesion associated with multiple neurofibromatosis.

In reviewing the present case, it is suggested that when removing a structurally similar pedunculated mass from the larynx, the base should be ligated or electrocoagulated.

SUMMARY.

1. A case of neurofibroma of the larynx associated with a generalized neurofibromatosis (Von Recklinghausen's disease) is presented.

2. Of the seven cases of neurofibroma of the larynx previously reported in the literature, only one was associated with the characteristic findings of Von Recklinghausen's disease.

3. Therapy consisted of snare and forceps removal of the laryngeal mass. A postoperative hemorrhage necessitated tracheotomy and pharyngeal packing.

4. There has been no evidence of local recurrence.

BIBLIOGRAPHY.

1. ANDERSON, W. A. D.: Pathology. St. Louis, Mo.: C. V. Mosby Co., pp. 1251-1252.
2. NEW, GORDON, and ERICH, JOHN B.: Benign Tumors of the Larynx. *Arch. Otolaryngol.*, 28:841, Dec., 1938.
3. VAN LOON, EMILY, and DIAMOND, SIDNEY: Neurofibroma of the Larynx. *Ann. Otolaryngol.*, 51:122-126, Mar., 1942.

NEW PRODUCT: RHINALGAN.

The Doho Chemical Corp., New York, N. Y., makers of Auralgan, Otosmosan and Rectalgan, introduce their new prescription product, Rhinalgan, the long-lasting nasal decongestant.

Rhinalgan is a colorless, nontoxic, bactericidal nasal decongestant containing: Desoxyephedrine Saccharinate 0.50 per cent w/v in an isotonic aqueous solution with 0.02 per cent Laurylammonium saccharin. Flavored. pH 6.4. It is efficient, long-lasting—with no systemic effect (pressor or respiratory). It will not cause rise in blood pressure, rapid pulse, wakefulness, restlessness, nervousness, smarting or stinging—or secondary vasodilation. Indications include: common cold, allergic and hypertrophic rhinitis, sinus infections; for pre- and post-operative shrinkage of nasal mucosa; as a diagnostic aid in office procedures. *Especially suitable for infants and children.* It is administered topically with Dohony Spray-O-Mizer once every three hours—or as indicated. Supplied: 30 grams (1 fl. oz.) in Dohony Spray-O-Mizer—also in pint bottles for doctor's office and hospital use.

MEETING OF GERMAN SOCIETY OF THROAT, NOSE AND EAR SPECIALISTS.

The annual meeting of the National Society of Throat, Nose and Ear Specialists will take place at Bad Kissingen under the direction of the president, Prof. Max Meyer, of Wurzburg, on May 24-27, 1950. The principal presentation will be "Mistakes and Dangers in the Treatment of Throat, Nose and Ear Diseases" by Prof. Marx, Wurzburg, with the collaboration of Prof. Moritz Weber, Ochsenfurth. American colleagues are heartily welcome as guests. Inquiries concerning invitations should be addressed to the secretary of the society, Prof. H. Frenzel, Geiststr 10, Gottingen, Germany.

UNIVERSITY OF MINNESOTA COURSE IN OTOLARYNGOLOGY.

The University of Minnesota announces the seventh biennial Continuation Course in Otolaryngology to be held June 26, 27, 28, 29, 30, 1950. This course is designed to bring to the practicing otolaryngologist the newer concepts and developments in the specialty. The course will be under the direction of Dr. Lawrence R. Boies and associates of the University Medical School. Dr. Fred A. Figi, Dr. Henry L. Williams and others of the Graduate School faculty will participate in the instruction. Guest lecturers will include Dr. Percy Ireland, Toronto; Dr. LeRoy Schall, Boston; Dr. Philip Meltzer, Boston; and Dr. John Shea, Memphis.

The fee for this course is \$50.00. The enrollment is limited. Application should be made at an early date to the Director, Center for Continuation Study, University of Minnesota, Minneapolis 14, Minn.

BOOK REVIEW.

Theory of Hearing. By Ernest Glen Wever, Professor of Psychology, Princeton University. 584 pages with index and 137 illustrations. New York, N. Y.: John Wiley and Sons, Inc., 1949. Price \$6.00.

This book is a technical monograph written by a psychologist for psychologists. It is a very personal book and sets forth the essence of the experimental work and theoretical interpretations by Prof. Wever and his collaborators over a period of 20 years. In it we find many details of the anatomy of the inner ear, and measurements of the size of many of the fine structures, and calculations based on these measurements that have not before been published. Here the book makes its most notable original contribution, and for this reason if for no other it will take its place among the classical treatments of the mechanism of hearing. Prof. Wever is undoubtedly the psychologist who is best acquainted with the anatomy of the ear. With his threefold experience in psychology, bio-acoustics and anatomy, he is able to make several unique contributions to our ideas of exactly how the ear receives sound waves and translates them into nerve impulses.

Prof. Wever's interest in anatomy is secondary to, and has its roots deep in his primary interest in auditory theory. His book is well named. Theories, not observations for their own sake and not practical applications, are his concern throughout. Prof. Wever and his colleague, Dr. Bray, are famous for their discovery of the electrical activity of the inner ear that is often known as the "Wever-and-Bray phenomenon," and they have put this phenomenon to good use as a tool wherewith to analyze many points about the bio-acoustics of the middle and the inner ear. Even their original experiment (placing an electrode in the auditory nerve of an anesthetized cat and "listening in" on the stream of nerve impulses going to the brain) was undertaken specifically to test a particular theory of hearing.

A central point in theories of hearing is the exact relationship between the pattern of nerve impulses and the pattern of the sound waves that stimulate them. Are the frequencies of sound waves carried to the brain as corresponding frequencies of nerve impulses? If so, does the brain make use of this information to recognize the "pitch" of a sound? If not, how does the ear analyze sounds in order to convey the information to the brain in some other form (such as sending the impulses over one set of nerve fibres rather than another)? The Wever and Bray experiment gave a partially affirmative answer to the first question. The frequency of the sound waves for low tones is in fact reproduced in the impulses in our auditory nerves. Prof. Wever has developed his theory of hearing, the "volley theory" as he calls it, to show that the brain might and probably does use these frequencies as an important clue for the recognition of pitch. He believes that his "volley principle" is important for all low tones and for medium tones up to almost the highest note on the piano.

If we can identify a plot or story in this book, we might call it "the rise and fall of the Helmholtz theory of hearing." Prof. Wever reviews in some detail the historical development of theories of hearing, and here again the book performs a useful service. Like Rosenblith and Békésy, who have also recently reviewed this subject, he makes it clear that the development of theories of hearing was limited at all points by the current knowledge of anatomy, physics and neurophysiology. The idea, formulated specifically by Helmholtz, that the newly recognized principle of "resonance" or sympathetic vibration might be the basis of a theory of hearing was fairly satisfactory in its day when less information was available than now. The principle of specific "tuning" by resonance was further developed by some of Helmholtz's followers, but it has few adherents today. Much of Prof. Wever's argument and many of his calculations are devoted to demolishing the remnants of the "Helmholtz" theory of specific isolated resonators in the inner ear. By the final chapter the reviewer had the impression that the author had been battling a straw man or perhaps a mummy, but Prof. Wever never relented!

Prof. Wever accepts the general present-day view that the inner ear does in fact analyze high-pitched sounds in a way somewhat like Helmholtz pictured, but for rather different reasons. The place or, better, the "place-pattern" of vibration in the inner ear is the essential feature. Some of us believe that the same general principle of place-pattern applies for all frequencies. Some may wonder why Prof. Wever passes so lightly over some of the evidence in favor of such an idea. The work of Stevens and Lurie, for example, is dismissed with hardly a word as apparently untrustworthy and not sufficiently detailed in presentation. The reviewer must protest this cavalier disregard of the work of his colleagues. In any case, Prof. Wever prefers a hybrid theory and invokes the frequency of nerve impulses and his "volley principle" as the chief cue for pitch for all tones below 1,000 cycles per second and as a partial cue for frequencies up to 4,000 cycles per second.

With Prof. Wever's final summarizing chapter the present reviewer can agree in full except for two or three sentences. The area of agreement far outweighs any difference of opinion on certain details such as the importance of the volley principle. Prof. Wever not only points out the difficulties of other theories, but he is also bold enough to formulate his own in full. In spite of some lack of clarity in the application of his theory to specific problems like tinnitus, diplacusis and binaural hearing, the main tenets of the theory are amply clear. Prof. Wever plays "doubles or quits." Within the next decade or two at the present rate of progress his theory may be as disregarded as that of Dr. Max Mayer, as outmoded as the formulations of Charles Bell, or so fully accepted that the rest of us will be saying that it is exactly what we believed all the time!

H. D.

FEBRUARY 1, 1950

**HEARING AIDS ACCEPTED BY THE COUNCIL ON
PHYSICAL MEDICINE OF THE
AMERICAN MEDICAL ASSOCIATION.**

As of February 1, 1950.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

**Micronic Model 101 (Magnetic Receiver); Micronic Model 303.
(See Silver Micronic.)**

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic (Crystal Receiver) Model 101; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 25-27, 1950.

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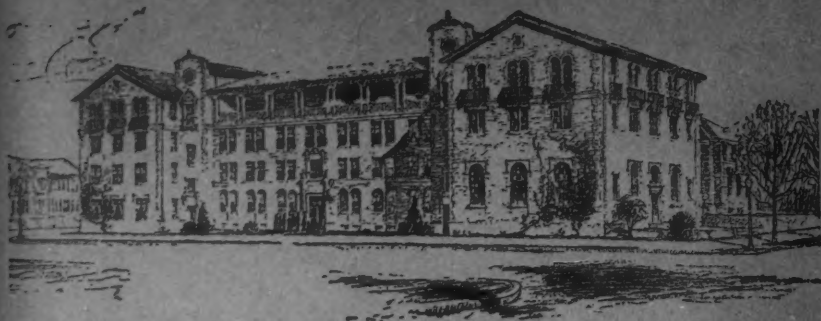
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